"A COMPARATIVE STUDY OF INJ.BUPIVACAINE 0.5% AND INJ.ROPIVACAINE 0.5% FOR SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK"

Submitted by

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In partial fulfillment of the requirements for the degree of

M. D.

in

ANAESTHESIOLOGY

Under the guidance of

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I hereby declare that this dissertation entitled **"A COMPARATIVE STUDY OF INJ.BUPIVACAINE 0.5% AND INJ.ROPIVACAINE 0.5% FOR SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK"** is a bonafide and genuine research work carried out by me under the guidance of **DR.VIJAY.V.KATTI** M.D., Associate Professor, Department of Anaesthesiology, at BLDE University's Shri B.M. Patil Medical College Hospital and Research Centre, Vijayapur.

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LIST OF ABBREVIATIONS

ASA	American Society of Anaesthesiologists
BP	Blood Pressure
BT	Bleeding time
CC	Cardiovascular collapse
CVS	Cardiovascular system
cms	Centimeters
CNS	Central nervous system
CT	Clotting time
DC	Differential count
ECG	Electrocardiogram
GA	General Anaesthesia
Hb	Hemoglobin
HR	Heart rate
I.P No	Inpatient number
IM	Intramuscular
IV	Intravenous
inj.	Injection
kg	Kilogram
L.A.	Local Anaesthetic
MAP	Mean arterial pressure
min	Minutes
mg	Milligram
ml	Milliliter
mm	Millimeter

mEq/L	Milli equivalent per liter
mg/dL	Milli gram per deci liter
mm Hg	Milli meter of mercury
MHz	Megahertz
NBM	Nil by mouth
NIBP	Non invasive blood pressure
PR	Pulse rate
P/A	Per abdomen
RS	Respiratory system
RR	Respiratory rate
RBS	Random blood sugar
S.D	Standard deviation
SpO_2	Oxygen saturation
TC	Total count
VAS	Visual Analogue Scale
yrs	Years

ABSTRACT

BACKGROUNDS AND OBJECTIVES

Brachial plexus block at the supraclavicular level provides safe, effective, low cost anaesthesia with excellent post operative analgesia. The current study was an attempt to compare the effect of Inj.Bupivacaine 0.5% and Inj.Ropivacaine 0.5% in supraclavicular brachial plexus block for patients undergoing upper limb orthopedic surgeries with respect to onset of sensory blockade, onset of motor blockade, duration of sensory blockade, duration of motor blockade and any adverse effects were noted.

MATERIALS AND METHODS

The Present study entitled "A comparative study of Inj.Bupivacaine 0.5% and Inj.Ropivacaine 0.5% for supraclavicular brachial plexus block" was carried out in the Department of Anaesthesiology, B.L.D.E. University's, Shri B.M. Patil Medical College Hospital and Research Centre, Vijayapur from October 2013 to June 2015.

Each patient was randomly allocated to one of the two groups of 39 patients each.

Group B – Bupivacaine group received 30 ml Bupivacaine 0.5% (5 mg/ml)

Group R – Ropivacaine group received 30 ml Ropivacaine 0.5% (5 mg/ml)

PARAMETERS

The effect was studied with respect to onset of sensory blockade, onset of motor blockade, duration of sensory blockade, duration of motor blockade and any adverse effects were noted.

RESULTS

Onset of sensory block :

In our study, we observed that onset of sensory block was earlier in Bupivacaine group (Group B) having a mean value of 16.6±3.2 minutes in comparison with Ropivacaine group (Group R) having a mean value of 19.9±4.0 minutes which was statistically significant.

Onset of motor block :

In our study, we observed that onset of motor block was earlier in Bupivacaine group (Group B) having a mean value of 21.4±2.6 minutes in comparison with Ropivacaine group (Group R) having a mean value of 25.9±2.4 minutes which was statistically significant.

Duration of sensory block :

In our study, we observed that duration of sensory block was longer in Bupivacaine group (Group B) having a mean value of 343.8±44.4 minutes in comparison with Ropivacaine group (Group R) having a mean value of 317.9±29.1 minutes which was statistically significant.

Duration of motor block :

In our study, we observed that duration of motor block was longer in Bupivacaine group (Group B) having a mean value of 387.4±36.0 minutes in comparison with Ropivacaine group (Group R) having a mean value of 368.7±33.1 minutes which was statistically significant.

Variations in blood pressure, heart rate, SpO_2, respiratory rate were statistically not significant in both the groups. No patient in our study developed any significant side effects.

CONCLUSION

Inj.Bupivacaine 0.5 % has early onset of sensory blockade, early onset of motor blockade, prolonged duration of sensory blockade, prolonged duration of motor blockade, when compared to Inj.Ropivacaine 0.5 % at equal volumes. Both the drugs maintain stable hemodynamic profile peri-operatively and are devoid of any adverse effects at the concentration and volumes used for the study. .

Keywords : Supraclavicular brachial plexus block; Bupivacaine; Ropivacaine.

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INTRODUCTION

Peripheral nerve blocks have assumed a prominent role in modern anaesthesia practice as they provide ideal operative conditions without any sedation or systemic hemodynamic effects. It has become an essential and growing part of anaesthesia. It offers an excellent alternative for patients who are haemodynamically compromised or too ill to tolerate general anaesthesia.

Orthopedic and plastic reconstructive surgeries can be of prolonged duration, hence adequate sensory and motor blockade along with profound analgesia are the main requirements for such surgeries. Regional anaesthesia offers various advantages over general anaesthesia particularly in emergency situations, where the patients are full stomach, not adequately starving and in high risk patients. It allows better postoperative recovery, good postoperative analgesia, preserves mental functions and prevents complications of intubation, laryngoscopy and general anaesthesia.

Regional nerve blocks are based on the concept that pain is conveyed by nerve fibres, which can be interrupted anywhere along their pathway. Local anaesthetics administered as regional nerve blocks provide post-operative pain relief by blocking signal transmission to dorsal horn.

Brachial plexus block is the preferred regional anaesthesia technique and a useful alternative to general anaesthesia for upper limb surgeries. They achieve near ideal operating conditions by producing complete muscular relaxation, maintaining stable intra-operative hemodynamics and decreases post-operative pain, vasospasm and edema.

Brachial plexus block at the supraclavicular level provides anaesthesia for the upper limb surgeries by blocking the middle & lower plexus (median nerve, radial nerve and ulnar nerve)

Elicitation of paraesthesia were traditionally used, now nerve locators and ultrasound technique are being used for proper nerve localization. Eliciting paraesthesia can be unpleasant and any slight sudden movement by the patient may displace the needle leading to failure of technique and occasionally neural damage. There is significant subjective variation in paraesthesia technique. Peripheral nerve locators aid in optimal needle placement thus minimizing unpleasant paraesthesia and also reducing any incidence of neural damage. Ultrasound guidance for peripheral nerve block provides a higher rate of block success, shorter procedure time and faster onset time. Ultrasound improves efficacy of peripheral nerve block compared with techniques that utilize peripheral nerve stimulator for nerve localization.

With advances in the field of surgery, surgical procedures have become more complex and the operating time has increased manifold with a consequent need to increase the duration of the brachial plexus block. Local anesthetic drugs have been traditionally used to provide anaesthesia and analgesia in regional nerve blocks.

In 1881, Carl Koller demonstrated ocular surface anaesthesia with Cocaine. Ester local anaesthetics developed later lost their value due to short duration of action, allergic reaction and systemic toxicity. Synthesis of Lignocaine in 1943 laid foundation for the studies of amide local anaesthetics. Various amide local anaesthetics like Mepivacaine, Prilocaine, Etidocaine and Bupivacaine have been used successfully.

Bupivacaine is a long acting local anaesthetic. Due to its long duration of action, combined with its high quality sensory blockade compared to motor blockade it has been the most commonly used local anaesthetic for peripheral nerve blocks.

Ropivacaine is a newer, long acting local anaesthetic whose neuronal blocking potential used in peripheral nerve blockade seems to be equal or superior to Bupivacaine.[1] Studies show that it has significantly greater safety margin over Bupivacaine because of lower CNS toxicity and cardiac toxicity and hence can be used in higher concentrations.[2] One of the drawbacks of Ropivacaine mentioned is its less intense motor blockade compared to Bupivacaine.[3]

Hence here is an attempt through the study to compare the effect of inj.Bupivacaine 0.5%, 30ml and inj. Ropivacaine 0.5%, 30ml in supraclavicular brachial plexus block for upper limb orthopedic surgeries using ultrasound guided approach.

AIMS AND OBJECTIVES OF THE STUDY

The present study was a randomized comparative study carried out in the Department of Anaesthesiology, B.L.D.E University's Shri B.M. Patil Medical College Hospital and Research Centre, Vijayapur with the objective to compare the effect of Inj.Bupivacaine 0.5% & Inj.Ropivacaine 0.5% in supraclavicular brachial plexus block for upper limb orthopedic surgeries with respect to:

- Onset of sensory blockade.
- Onset of motor blockade.
- Duration of sensory blockade.
- Duration of motor blockade.
- Any adverse effects.

REVIEW OF LITERATURE

Pain is the mechanism for informing an organism of a dangerous situation. In ancient day's opium, hashish, alcohols were used to reduce the pain. The International association for the Study of Pain (IASP) defines pain as an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage.

In 1884, Halsted performed the first brachial plexus nerve block when he found the cords and nerves of the brachial plexus, after blocking the roots in the neck with cocaine solution 0.1% under direct vision.

In January 1900, Harvey Cushing (1869–1939), who was at that time one of Halsted's surgical residents applied cocaine to the brachial plexus prior to dividing it, during a forequarter amputation for sarcoma.

The first percutaneous supraclavicular block was performed in 1911 by German surgeon Diedrich Kulenkampff (1880–1967).[4] Kulenkampff subjected himself to the supraclavicular block in what is known as Classical approach. Subsequently, studies showed a high incidence of pneumothorax (2-6%) with this approach and modifications of supraclavicular technique have been developed in an effort to decrease incidence of pneumothorax. In 1917 infraclavicular approaches to the brachial plexus were described by L.Bazy and V.Pauchet and later in 1973 popularized by P.Raj.[5]

George Hirschel (1875–1963) later in the same year described a percutaneous approach to the brachial plexus from the axilla. He made separate injections above and below the axillary artery with a four inch needle directed towards the apex of axilla.[6]

In 1928, Kulenkampff and Persky published their experiences with a thousand blocks without apparent major complications.[7] They described their technique with the patient in the sitting position or in the supine position with a pillow between the shoulders. The needle was inserted above the midpoint of the clavicle where the pulse of the subclavian artery could be felt and it was directed medially towards the second or third thoracic spinous process.

The subclavian (supraclavicular) perivascular technique was first developed by Alon Willie and Collins.[8] They reported less than 1% incidence of pneumothorax. Patrick in 1940 published his modification of Kulenkamff technique.[9]

In 1927, Gaston Labat first described the interscalene approach but the modern use of this technique is due to Alon Willie.[10]

In 1967, Macintosh and Mushin described the supraclavicular block which involved blocking the plexus as it lies on the first rib lateral to the subclavian artery. This approach had higher success rate and fewer complications.[11]

In 1976, James K Sims modified the infraclavicular approach. He inserted the needle inferior and medial to clavicle and proved that this approach decreased the incidence of pneumothorax and was easier to perform.[12]

In 1994, Fleck and Moorthy compared supraclavicular, lateral paravascular and axillary approaches. The lateral paravascular approach is safer but success rate of only 72%, with the necessity of nerve stimulation and doppler probe to perform the technique.[13]

In 1912, Von Perthes used special needles covered with insulation or shielding for nerve stimulation for regional blockade. He described the use of an induction apparatus using a faradic current, which was then transmitted down a pure nickel needle previously coated with lacquer down to the tip to provide insulation. With the advent of nerve stimulators in nineteenth century, blocks were being performed with the aid of nerve locator.

Saranoff in 1950 and 1951 and Pearson in 1955 located motor nerves by electrical stimulation with an insulated needle.[14]

In 1984, Pither, Raj and Ford demonstrated the use of peripheral nerve stimulator for regional anaesthesia.

Peripheral nerve blocks have become important in clinical practice because of their role in post operative pain relief, shortening of patient recovery time & avoiding adverse effects of general anaesthesia.[15] Different means have been used to shorten the delay in onset of action, improve the degree of blockade and duration of action.

Bupivacaine is a long acting local anaesthetic. Due to its long duration of action, combined with its high quality sensory anaesthesia relative to motor blockade it has been the most commonly used local anaesthetic for peripheral nerve blocks.

Ropivacaine is a newer long acting local anaesthetic whose neuronal blocking potential used in peripheral nerve blockade seems to be equal or superior to Bupivacaine.[1] Studies show that it has significantly greater safety margin over Bupivacaine because of lower central nervous system and cardiovascular toxicity.[2]

Ropivacaine is safer than Bupivacaine as it has lower CNS toxicity and cardiac toxicity. Thus it would be the preferred local anesthetic for brachial plexus blockade when long-lasting anesthesia and analgesia is required.

Hickey *et al,*[16] in 1991 conducted study titled "A comparison of Ropivacaine 0.5% and Bupivacaine 0.5% for brachial plexus block". Forty-eight patients received a subclavian perivascular brachial plexus block for upper-extremity surgery. One group (n = 24) received Ropivacaine 0.5% (175 mg) and a second group (n = 24) received Bupivacaine 0.5% (175 mg), both without epinephrine. Onset times for analgesia and anesthesia in each of the C5 through T1 brachial plexus dermatomes did not differ significantly between groups. Duration of analgesia and anesthesia were long (mean duration of analgesia, 13-14 h; mean duration of anesthesia, 9-11 h) and also did not differ significantly between groups. Motor block was profound, with shoulder paralysis as well as hand paresis developing in all of the patients in both groups. Two patients in each group required supplemental blocks before surgery. Ropivacaine 0.5% and Bupivacaine 0.5% appeared equally effective in providing brachial plexus anesthesia.

Hickey *et al,*[17] in 1992 conducted a study titled "A comparative study of 0.25% Ropivacaine and 0.25% Bupivacaine for brachial plexus block". In view of the frequent need for supplementation noted with both 0.25% Ropivacaine and 0.25% Bupivacaine, the study did not recommend using the 0.25% concentrations of these local anesthetics to provide brachial plexus block.

Narendra Babu *et al,*[18] in 2014 conducted a study titled "A comparative study of Bupivacaine 0.5% and Ropivacaine 0.5% for supraclavicular brachial plexus block (perivascular approach)". Sixty patients received a subclavian perivascular brachial plexus block for elective upper limb surgeries after getting ethical clearance. One group (n = 30) received 30ml Bupivacaine 0.5% and a second group (n = 30) received 30ml Ropivacaine 0.5%. The mean onset time of sensory block was earlier in group

B, 17.70±2.35 minutes in comparison with group R, 22.13±0.5 minutes ($p<0.05$). The mean onset time of motor block was earlier in group B, 25.43±2.22 minutes in comparison with group R, 27.90±1.88 ($p<0.05$). Duration of sensory blockade was prolonged in group B, 342.00±47.66 minutes in comparison with group R, 302.00±42.38 minutes ($p<0.05$). Duration of motor blockade was prolonged in group B, 369.00±41.05 minutes in comparison with group R, 336.00±37.29 minutes ($p<0.05$) Both drugs are devoid of any adverse effects with the concentration and volume used.

Vainionpaa *et al,*[19] in 1995 compared 0.5% Ropivacaine with 0.5% Bupivacaine in axillary brachial plexus block and found no statistically significant differences in the clinical and pharmacokinetic comparisons. They used a slightly different dose of drug depending on patient body weight: 30 ml (weight <70 kg), 35 ml (weight 70–80 kg) or 40 ml (weight >80). The median onset times for anaesthesia and complete motor block were in the range of 12-48 minutes and 5-20 minutes respectively. Thirty-eight percent of patients in the Ropivacaine group and 29% in the Bupivacaine group needed additional nerve block(s) or supplementary analgesia and 7% in the Bupivacaine group needed general anesthesia for surgery. Anesthesia was achieved in 52% - 86% of the evaluated six nerves in the Ropivacaine group and in 36% - 87% in the Bupivacaine group; the lowest figures were seen in the musculocutaneous nerve. In the pharmacokinetic study the mean peak plasma concentrations (C_{max}) were 1.28+/- 0.21 mg/l in the Ropivacaine group and 1.28+/- 0.47 mg/l in the Bupivacaine group and median times to peak plasma concentration (t max) were 0.86 hours and 0.96 hours respectively. Median terminal half-lives (t 1/2) were 7.1 hours and 11.5 hours in the Ropivacaine group and Bupivacaine group, respectively (P = 0.07).

Klein SM *et al,*[20] in 1998 conducted a study titled "A comparison of 0.5% Bupivacaine, 0.5% Ropivacaine, and 0.75% Ropivacaine for interscalene brachial plexus block". Patients were assigned (n=25 per group) to receive an interscalene block using 30 ml of 0.5% Bupivacaine, 0.5% Ropivacaine, or 0.75% Ropivacaine. At 1-min intervals after local anesthetic injection, patients were assessed to determine loss of shoulder abduction and loss of pinprick in the C5-6 dermatomes. Before discharge, patients were asked to document the time of first oral narcotic use, when incisional discomfort began, and when full sensation returned to the shoulder. The mean onset time of both motor and sensory blockade was <6 min in all groups. Duration of sensory blockade was similar in all groups as defined by the three recovery measures. They concluded that there is no clinically important difference in times to onset and recovery of interscalene block for Bupivacaine 0.5%, Ropivacaine 0.5%, and Ropivacaine 0.75% when injected in equal volumes.

McGlade *et al,*[21] in 1998 conducted "A comparative study of 0.5% Ropivacaine and 0.5% Bupivacaine for brachial plexus block". The purpose of this study was to compare the use of 0.5% Ropivacaine with 0.5% Bupivacaine for axillary brachial plexus anaesthesia. Sixty-six patients undergoing upper limb surgery were enrolled in a double-blind, randomized, multicentre trial. Five patients were subsequently excluded for various reasons. Of the remaining patients, 30 received 40 ml of 0.5% Ropivacaine and 31 received 40 ml of 0.5% Bupivacaine. Brachial plexus block was performed by the axillary approach using a standardized technique with a peripheral nerve stimulator. Parameters investigated included the frequency, onset and duration of sensory and motor block, the quality of anaesthesia and the occurrence of any adverse events. The six principal nerves of the brachial plexus were studied individually. The frequency for achieving anaesthesia per nerve ranged from

70 to 90% in the Ropivacaine group and 81 to 87% in the Bupivacaine group. The median onset time for anaesthesia was 10 to 20 minutes with Ropivacaine and 10 to 30 minutes with Bupivacaine, and the median duration was 5.3 to 8.7 hours with Ropivacaine and 6.9 to 20.3 hours with Bupivacaine. Motor block was evaluated at the elbow, wrist and hand, and was completely achieved at a rate of 60 to 73% in the Ropivacaine group and 55 to 71% in the Bupivacaine group. The median duration of motor block was 6.5 to 7.5 hours with Ropivacaine and 6.0 to 9.0 hours with Bupivacaine. These parameters were not statistically different. The duration of partial motor block at the wrist (6.8 v 16.4 hours) and hand (6.7 v 12.3 hours) was significantly longer with Bupivacaine. Ropivacaine 0.5% and Bupivacaine 0.5% appeared equally efficacious as long-acting local anaesthetics for axillary brachial plexus block.

Vaghadia *et al,*[22] in 1999 conducted "A study to compare efficacy of 30 ml of Ropivacaine 7.5mg/ml with 30 ml Bupivacaine 5mg/ml for supraclavicular brachial plexus anesthesia". After taking informed consent, 104 ASA I-III adults participated in a randomized, double-blind, multi-center trial to receive 30ml of either Ropivacine 7.5mg/ml or Bupivacaine 5mg/ml for subclavian perivascular brachial plexus block. Onset and duration of sensory and motor block were assessed. Onset times and duration of sensory and motor block were similar between groups. Mean duration of analgesia was between 11.3 and 14.3 hours with Ropivacaine and between 10.3 and 17.1 hours with Bupivacaine. Quality of muscle relaxation judged as excellent by the investigators was not significantly different between two groups. One patient developed a grand mal seizure shortly after receiving Bupivacaine and recovered consciousness within 30 minutes with no residual effects. There were no adverse effects in the Ropivacine group. This suggests that lower CNS and cardiotoxicity of

Ropivacaine reduces the risk to the patient due to inadvertent intravenous injection. They concluded that 30 ml Ropivacaine 7.5 mg/ml or 30 ml Bupivacaine 5mg/ml produced satisfactory and comparable sensory and motor block.

Bertini *et al,*[23] in 1999 conducted a study "axillary brachial plexus block with 32 ml of 0.5% Ropivacaine, 0.75% Ropivacaine and 0.5% Bupivacaine by a nerve stimulator technique". They found similar efficacy between equal concentrations of Bupivacaine and Ropivacaine, with no improvement of onset time or duration of action when comparing 0.5% Bupivacaine with 0.75% Ropivacaine. Although onset time with Ropivacaine was faster than with Bupivacaine, the mean onset time of sensory blockade was >16 minutes for axillary plexus block in their study, and the quality of anaesthesia was better with Ropivacaine. The rate of complete sensory and motor block observed with both Ropivacaine groups was higher at 10, 15, and 20 minutes post injection ($P < 0.001$). The mean peak time was shorter with Ropivacaine than with Bupivacaine (R50 = 16.37 minutes, R75 = 14.7 minutes, B = 22.3 minutes, $P < 0.05$). Because no statistical differences were found between two Ropivacaine groups, they concluded that 0.75% does not add benefit and that 0.5% Ropivacaine should be used to perform axillary brachial plexus blocks.

Reader *et al,*[24] in 1999, in their study "Axillary brachial plexus block with Ropivacaine 7.5 mg/dl, a comparative study with Bupivacaine 5mg/ml" showed that 0.75% Ropivacaine used for axillary brachial block resulted in better anaesthesia when compared with same volume of 0.5% Bupivacaine, however the onset and duration of blockade were similar in both groups and concluded that Ropivacaine at a concentration of 7.5 mg/ml was required to produce similar effects with respect to onset and duration of sensory and motor blockade as compared to Bupivacaine 0.5 % at equal volumes.

Casati *et al,*[25] in 2000 studied 0.5% Ropivacaine or 0.5% Bupivacaine in interscalene brachial plexus. Thirty ASA I and II patients scheduled for elective shoulder surgery by using nerve stimulator. They concluded that 0.5% Ropivacaine has long duration in postoperative pain relief and lower potential for central nervous system and cardiovascular toxicity.

McClellan *et al,*[26] in 2000 conducted a study titled - Ropivacaine "An update of its use in regional anaesthesia". They concluded that Ropivacaine is a well tolerated regional anaesthetic with an efficacy broadly similar to that of Bupivacaine. However, it may be a preferred option because of its reduced central nervous system and cardiotoxic potential and its lower propensity for motor block.

Borgeat *et al,*[27] in 2001 compared the effects of patient controlled interscalene analgesia with 0.2% Ropivacaine and 0.15% Bupivacaine on handgrip strength after major open shoulder surgery and concluded that for similar pain control, Ropivacaine was associated with better preservation of strength in hand and less parasthesia in fingers.

Singelyn *et al,*[2] in 2001 conducted a study titled "Clinical application of Ropivacaine for the upper extremity"**.** This review demonstrates that Ropivacaine is effective in brachial plexus anesthesia. It is at least as efficient as Bupivacaine in terms of quality, duration of analgesia, anesthesia, and motor block. It could have some advantages over Bupivacaine in terms of onset time of sensory and motor block, but this remains controversial. In single-shot brachial plexus block, it is equipotent to Bupivacaine and has a similar pharmacokinetic profile. Its minimal effective concentration is 0.5%, and the benefit of increasing its concentration to 0.75 or 1% remains debatable.

Tripathi *et al,*[28] in 2012 conducted a study titled "Supraclavicalar brachial Plexus block for upper limb orthopedic Surgery: A Randomized, double blinded comparison between Ropivacaine And Bupivacaine" . The mean onset time of motor block was 8.92 ± 2.92 min and 15.86 ± 3.72 min (P<0.05), peak developed in 27.26 ± 8.93 minutes and 23.43 ± 3.89 min (P<0.05) and duration of 8.53 ± 1.02 hours and 8.77 ± 0.75 hours (P>0.05) in group R and group B respectively. In comparison to equal volume of 0.5% Bupivacaine, 0.75% Ropivacaine provides earlier onset and peak of sensory blockade (p<0.05) with comparable duration of postoperative analgesia (P>0.05). Though, it provides earlier onset of motor blockade (p<0.05), there is statistically significant delay in achieving peak effect as compared to Bupivacaine (p<0.05). Hemodynamics remained stable and no complications were encountered in both the groups.

Tsui *et al,*[29] in 2008 conducted a case series on ultrasound guided supraclavicular block using a curvilinear probe in 104 day-case hand surgery patients and reported successful experience using ultrasound guidance and nerve stimulation during supraclavicular blockade. They concluded that the curvilinear probe enables a large field of view, adequate resolution in larger patients, and excellent needle visibility that allows access to the plexus while avoiding the pleura and subclavian artery.

Mehta *et al,*[30] in 2015 conducted a study titled "Comparative study of supraclavicular brachial plexus block by nerve stimulator versus ultrasound guided method". The study was conducted in 50 patients and divided into group A (n=25) and group B (n=25). Group A was given block with nerve stimulator and group B was given ultrasound guided block. Duration of block performance was 10±2.5 minutes

in group A when compared to 6±1.5 minutes in group B. Mean onset time of sensory block was 9.64±1.14 minutes in group A when compared to 6.64±0.89 minutes in group B. Mean onset time of motor block was 12.18±1.48 minutes in group A when compared to 10.10±1.14 minutes in group B. In group B 15-25 ml of drug was required for successful block as compared to 20-35 ml for group A. One patient in group A has post operative pneumothorax and 5 patients required supplemental general anaesthesia as compared to no complications in group B and 2 patients required general anesthesia due to inadequate block in group B. They concluded that supraclavicular brachial plexus block using ultrasound guided method is an improved nerve block technique due to visualization of nerves with more success, decreased complication rate, faster onset and less time consuming as compared to nerve stimulator method but requires knowledge of sonoanatomy and skill to operate ultrasound machine.

Singh *et al,*[31] in 2014 conducted a study titled "Comparison between conventional technique and ultrasound guided supraclavicular brachial plexus block in upper limb surgeries. Of the 60 patients included in the study they were divided into two groups of 30 patients each. Group 1 received ultrasound guided supraclavicular brachial plexus block. Group 2 received conventional supraclavicular brachial plexus block. Time taken for the procedure to administer a block in group 2 was 5.43 minutes where as using an ultrasound, time required for the same was 10.1 minutes($P<0.0001$). Onset of sensory block was 10.86±3.19 minutes in group 1 as compared to 11.6±2.45 minutes in group 2 ($P=0.32$). Onset of motor block was 14.56±3.85 minutes in group 1 as compared to 16.8±3.42 minutes in group 2($P=0.02$). Duration of sensory block was 397.9±67.3 minutes in group 1 as compared to 352.22±87.5 minutes in group 2($P=0.03$).Duration of motor block was 343.44±60.8

minutes in group 1 as compared to 305.19±60.1 minutes in group 2 (P=0.02). Block was successful in 90% in group 1 and 77.3% in group 2. Incidence of vessel puncture was 10% in group 2 compared to 3.33% in group 1(P=0.05). They concluded that ultrasound guided supraclavicular brachial plexus block has more success rate and very few complications compared to block performed by conventional approach. Time taken for the block performed by ultrasound was longer than the conventional technique. Onset of sensory and motor blockade was little earlier by ultrasound technique. Duration of sensory and motor blockade was longer by ultrasound technique.

Hanumanthaiah *et al,*[32] in 2013 conducted a study on ultrasound guided supraclavicular block and they concluded that recent renewed interest in ultrasound guided supraclavicular blocks may be due to easy image acquisition relating to superficial location of the brachial plexus at this level and identifying the pleura thus minimizing the risk of pneumothorax.

Rupera *et al,*[33] in 2013 conducted a study titled "Ultrasonography guided technique offer advantage over peripheral nerve stimulator guided technique in supraclavicular brachial plexus block". The study was conducted among 60 patients suffering from chronic renal failure with ASA III scheduled for the creation of arterial-venous fistula. In group A (n=30) ultrasonography guided technique was used and in group B (n=30) peripheral nerve stimulation technique was used. Procedure time in group A was 4.55±0.74 minutes as compared to 5.71±0.92 minutes in group B (p<0.0001). Onset time for sensory block in group A was 2.97±0.72 minutes as compared to 3.63±0.76 minutes in group B (p=0.002). Onset time for motor block in group A was 4.55±0.78 minutes as compared to 5.13±0.71 minutes in group B (p=0.007). Time to achieve complete block in group A was 13.17±1.54 minutes as

compared to 16.96±1.83 minutes in group B ($p<0.0001$). Duration of sensory block in group A was 5.29±0.82 hours as compared to 4.73±0.81 hours in group B ($p=0.015$). Duration of motor block was 5.05±0.67 hours in group A as compared to 4.58±0.73 hours in group B ($P=0.02$). No patients in group A had any complications while in group B, 3 patients had subclavian artery puncture and 1 had pneumothorax ($p<0.05$). They concluded that ultrasonography guided supraclavicular brachial plexus block is quick to perform, offers improved safety and accuracy in identifying the position of nerves to be blocked and of the structures. Wider availability of USG is likely to ensure even greater use in the future and will become gold standard for peripheral nerve blocks over the more conventional techniques.

Vincent *et al,*[34] in 2003 conducted a study on ultrasound guided supraclavicular brachial plexus block. Forty healthy outpatients received ultrasound-guided supraclavicular brachial plexus blocks for elective upper limb surgery. For the first 29 patients, a Toshiba Core Vision Pro unit equipped with a linear 8-MHz probe was used. For the remaining 11 patients, a Philips ATL HDI 5000 SonoCT unit equipped with a linear 5-12 MHz probe, color Doppler, and compound imaging capability was used. They concluded that ultrasound guidance is clinically useful for supraclavicular brachial plexus block. It confers confidence and accuracy of needle placement for nerve localization and examines the pattern of local anesthetic spread.

ANATOMY OF BRACHIAL PLEXUS[35,36]

The brachial plexus supplies all of the motor and almost all of the sensory function of the upper extremity. The remaining area, the skin over shoulder is supplied by the descending branches of cervical plexus, and posterior medial aspect of arm, extending nearly to the elbow is supplied by medial cutaneous nerve of the arm and the intercostobrachial branch of second intercostal nerve.

Brachial Plexus is formed from the anterior primary rami of 5th, 6th, 7th and 8th cervical and 1st thoracic nerve and frequently receives small contributing branches from the fourth cervical and second thoracic nerve. After these nerves leave their respective intervertebral foramina, they proceed anterolaterally and caudally to occupy the interval between the anterior and middle scalene muscle, where they unite to form three trunks, thus initiating the formation of proper plexus. These trunks emerge from the interscalene space at the lower border of these muscles and continue anterolaterally and inferiorly to converge towards the upper surface of first rib, where they are closely grouped cephaloposterior to the subclavian artery.

At the lateral edge of the first rib, each trunk divides into an anterior and posterior division, each of which passes inferior to the mid portion of clavicle to enter the axilla through its apex. These divisions by which fibers of the trunk reassemble to gain the ventral and dorsal aspects of the limb reunite within the axilla to form three cords the lateral, medial and posterior named because of their relationship with the second part of axillary artery.

At the lateral border of pectoralis minor, the three cords break up to give rise to peripheral nerves of the upper extremity.

The lateral cord gives off the lateral head of median nerve, lateral pectoral nerve and musculocutaneous nerve.

The medial cord gives off the medial head of median nerve, medial cutaneous nerve of arm, medial cutaneous nerve of forearm, medial pectoral nerve and ulnar nerve.

The posterior cord gives off the upper and lower subscapular nerve, nerve to latissimus dorsi, radial nerve and axillary nerve (circumflex nerve).

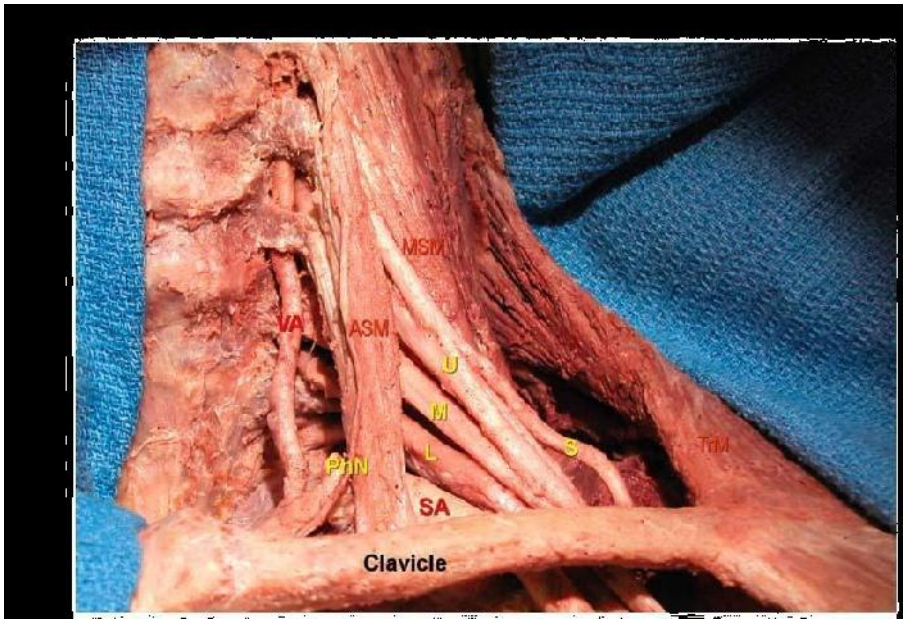
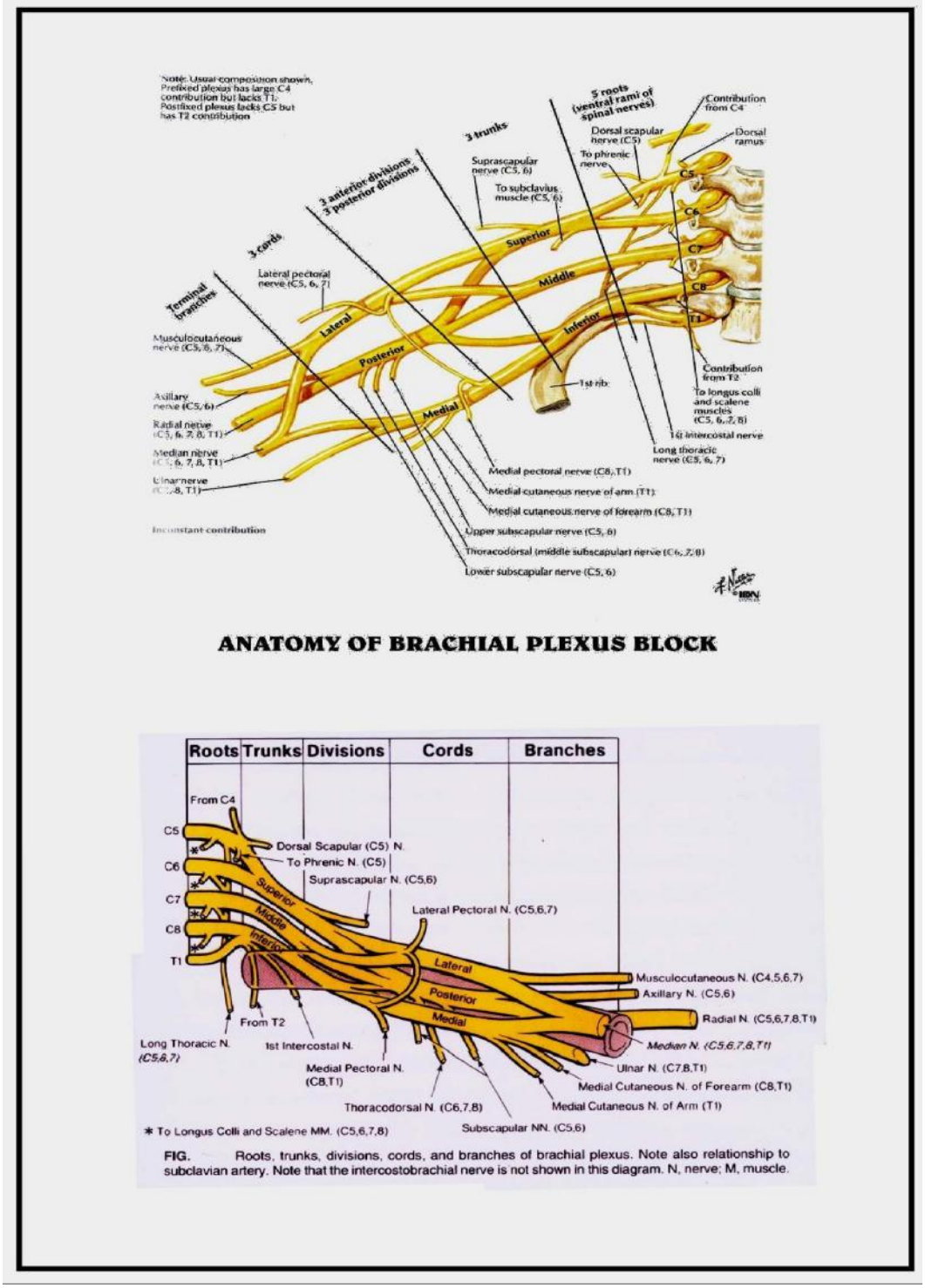


Figure - 1: Anatomy of brachial plexus

(ASM - Anterior scalene muscle, MSM - Middle scalene muscle, SA- Subclavian artery, PhN - Phrenic nerve)



ANATOMY OF BRACHIAL PLEXUS BLOCK

FIG. Roots, trunks, divisions, cords, and branches of brachial plexus. Note also relationship to subclavian artery. Note that the intercostobrachial nerve is not shown in this diagram. N, nerve; M, muscle.

Figure-2: Formation and branches of brachial plexus.

Livingstone and Werthein originally pointed out, and Winnie has refocused our attention on, the fascial barriers that surround these structures. The prevertebral fascia divides to invest the anterior and middle scalene muscles and then fuses at the lateral margins to form an enclosed interscalene space. Therefore, as the nerve roots leave the transverse processes, they emerge between the fascia that covers the anterior and middle scalene muscle, and in their descent toward first rib to form trunk of the plexus, the roots may be considered sandwiched between anterior and middle scalene muscles, the fascia of which serves as "Sheath" of plexuses. As the root passes through this space they converge to form the trunk of the brachial plexus and together with subclavian artery, invaginate the scalene fascia which form subclavian perivascular sheath. This, in turn, becomes the axillary sheath as it passes under the clavicle.

Branches from roots :

The nerve (of Bell) to the serratus anterior from C5, C6 and C7.

Dorsalis scapulae nerve from C5.

Muscular branches to the longus cervicis (C5-C8)

The three scalene (C5-C8), the rhomboids (C5)

Branch to the phrenic nerve (C5).

Branches from trunks :

Suprascapular nerve (C5 and C6)

Nerves to subclavius (C5 and C6).

Branches from cords :

From lateral cord	Lateral pectoral (C5-C7)
	Lateral head of the Median (C5-C7)
	Musculocutaneous (C5, C6, C7)
From posterior cord	Upper & lower subscapular (C5 & C6)
	Thoracodorsal nerve to the lattissimus dorsi (C5, C6, C7).
	Axillary (C5 and C6)
	Radial (C5, C6, C7, C8, and T1)
From Medial cord	Medial head of median (C8, T1)
	Medial pectoral (C8, T1)
	Medial cutaneous of the forearm (C8, T1)
	Medial cutaneous of the arm (T1)
	Ulnar (C8, T1)

Approaches to Brachial plexus block

(1) Interscalene approach.

(2) Supraclavicular approach.

(3) Infraclavicular approach.

(4) Axillary approach.

RELATIONS OF BRACHIAL PLEXUS

In its passage from the cervical transverse processes to the first rib, the plexus is “sandwiched” between the anterior and middle scalene muscles and invested in the fascia of those two muscles.

The ‘interfascial compartment’ along the subclavian artery which crosses the first rib immediately in front of the trunks. Artery is close to the scalenus anterior and the plexus close to the scalenus medius.

Subclavian vein is separated from the artery by the scalenus anterior. The fascia covering the muscles is derived from the prevertebral fascia, which splits to invest these muscles and rejoins again at their lateral margins to form an enclosed space, the interscalene space. As the plexus cross the first rib, the three trunks are stacked one on top of the other vertically. Not infrequently, the inferior trunk gets trapped behind and even beneath the subclavain artery above the rib, during embryologic development.

This may be reason why local anaesthetics injected via the interscalene technique sometimes fail to provide anaesthesia in the distribution of the ulnar nerve, which may be buried deep within inferior trunk behind or beneath the subclavian artery. After crossing the first rib, they split to form two divisions and the cords and subclavian artery becomes axillary artery. Above the clavicle, the axillary artery lies central to the three cords, in the axilla the lateral and posterior cords are lateral to the first of the axillary artery, the medial cord being behind it. Around the second part of the artery , they are related according to their names. In the lower axilla, cords divide into nerves for the upperlimb. In passing over the first rib under the clavicle, the subclavian vein also becomes the axillary vein and its relationship with the neurovascular bundle changes.

Above the first rib the subclavian vein doesn't lie within the neurovascular bundle, it is separated by the insertion of scalenus anterior. As it passes over the first rib, becoming the axillary vein it joins the neurovascular bundle, so that parts of the plexus are sandwiched between artery and vein. As all the three enter the axilla, they invaginate the perivertebral fascia at the lateral margins of the anterior and medial scalene muscles, carrying this fascial investment of the perivertebral or scalene fascia forming the axillary perivascular space, a tubular extension of the interscalene space. In its course through the axilla and upper arm the fascia of the surrounding muscles contribute to the axillary sheath, making it thick and tough, providing the fascial click to the anaesthetic while entering the sheath. It is important to note the major terminal nerves leave the sheath high in the axilla under cover of pectoralis minor muscle.

The musculocutaneous nerve enters the substance of coracobrachialis and continues down within the muscle. The axillary nerve also leaves the sheath immediately after arising from the posterior cord. The intercostobrachial nerve travels parallel to but outside the axillary sheath and medial cutaneous nerve of the arm runs similarly but occasionally it may remain within the sheath

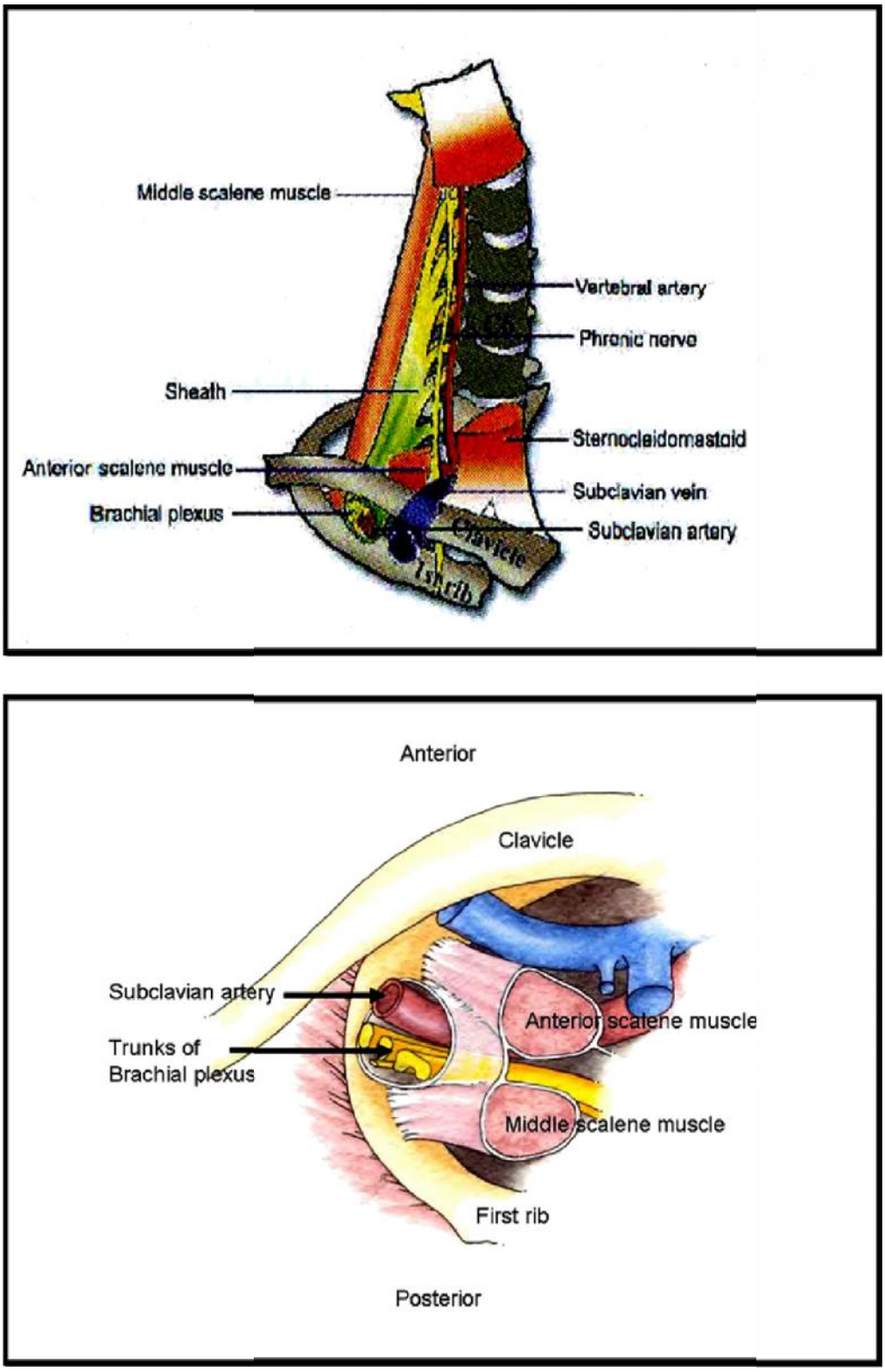


Figure 3: Brachial Plexus viewed from above

THE BRACHIAL PLEXUS SHEATH

Volume of the sheath : 42 ml.

Shape of the sheath : cylindrical to conical – wide proximally and narrow distally.

Length : 8- 10 cms long.

- The connective tissue of the prevertebral fascia and the anterior and middle scalene envelopes the brachial plexus as well as the subclavian and axillary artery in a neurovascular "sheath".
- The tissue is densely organized as it leaves the deep cervical fascia proximally, but becomes more loosely arranged distally. The sheath blends with the fascia of the biceps and brachialis muscle distally.
- Anatomic dissection, histologic examination and CT scanning after injection of radiocontrast into the sheath demonstrate the existence of connective tissue septae which extend inward from the fascia surrounding the sheath. The thin connective tissue septae frequently adhere to nerves and vessels leaving no free space between layers and compartmentalizing the components of the sheath.

Anesthetic implications:

Because of these connective tissue septae, anaesthesia might be complete and rapid in onset in some nerves, but delayed and incomplete or completely absent in others. The incidence of partial block is an exception rather than the rule, so septa apparently are of little clinical significance as the local anaesthetic can percolate through them.

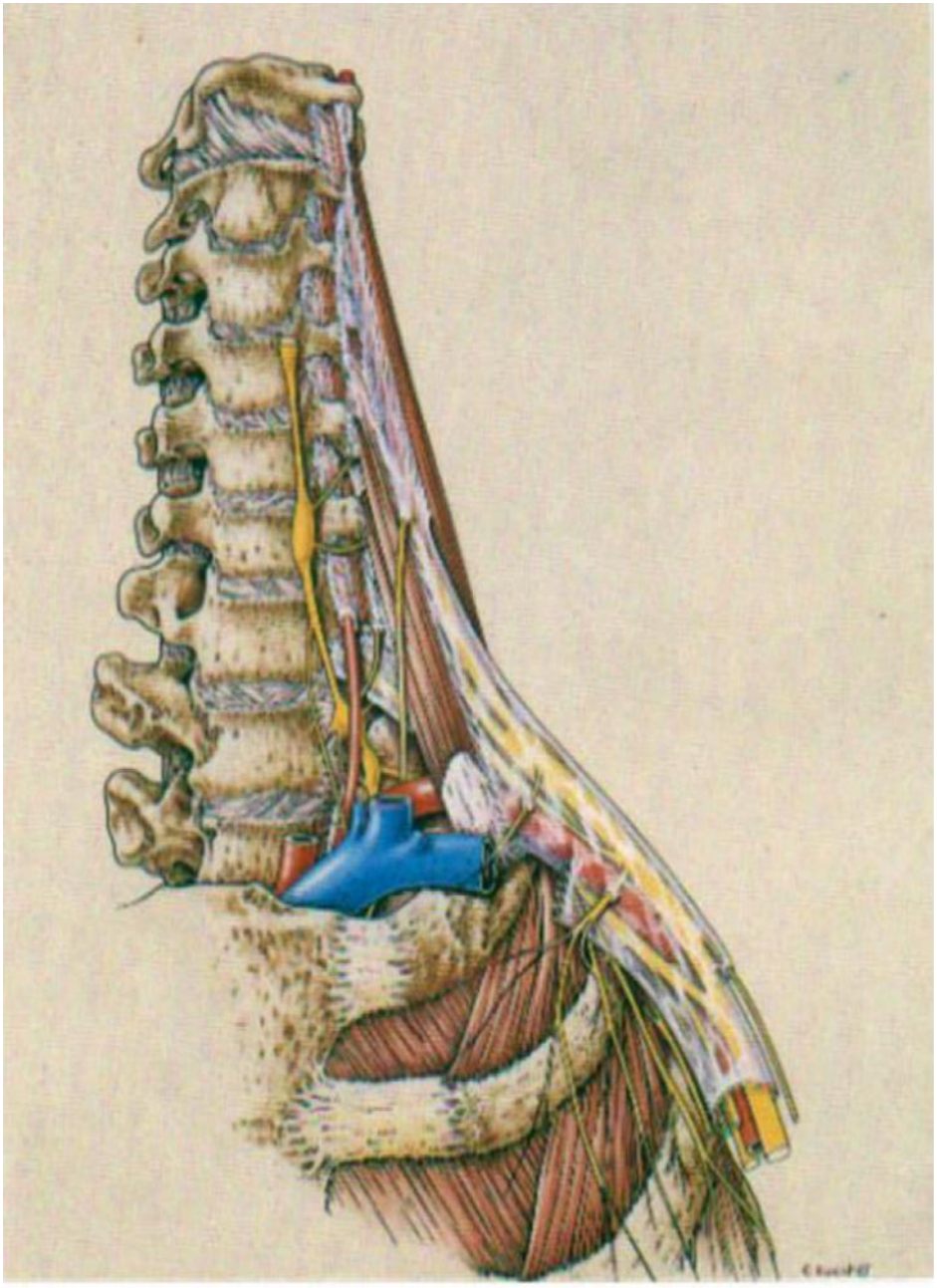


Figure- 4: Sheath around the brachial plexus.

SONOANATOMY OF BRACHIAL PLEXUS [37]

The brachial plexus in supraclavicular area is scanned using a high frequency 5-14 MHz linear ultrasound probe held in an oblique plane, (coronal or sagittal) which scans both in longitudinal and horizontal direction.

The subclavian artery is a prominent landmark identified immediately superior to first rib as a pulsatile hypoechoic tennis ball like image on ultrasound. The first rib appears as a bright hyperechoic rim with a drop out bony acoustic shadow. The brachial plexus normally appears superior, supero-lateral or superomedial to subclavian artery as multiple hypoechoic ovals/circles, often described as a honeycomb pattern or “bunch of grapes”. The brachial plexus may acquire a triangular, linear or vertical (or oblique) arrangement of trunks/division/cords around subclavian artery in supraclavicular region on ultrasound scan.

The pleura is seen as a hyperechoic line at same level as more “shiny” than the rib. Pleura moves and shines more with breathing. The hyperechoic pleural shadow does not have a drop out acoustic shadow, which differentiates it from the rib shadow. The scalenus anterior and medius muscles appear as hypoechoic structures on ultrasound scan and can be followed commencing from their origin to the point of insertion on first rib. The phrenic nerve lies on anterior surface of scalenus anterior from C4-7 level in neck. The long thoracic and dorsal scapular nerves pass through middle scalene muscle and may appear as “holes” or hypoechoic structures. Often part of brachial plexus passes through scalenus anterior or medius muscles and is seen as small round or oval hyperechoic or hypoechoic structures.

The thyrocervical trunk and transverse cervical artery often appear similar to nerve trunks on ultrasound scan. The pulsations of smaller arteries or branches are easily masked by the strong pulsations of subclavian artery. These vessels may fall in nerve block needle trajectory or course along or through the brachial plexus. This poses a threat of vascular injury, hematoma formation or inadvertent intra-vascular injection. Color Doppler may help in differentiation of brachial plexus from arteries by demonstrating color enhancement. Thus ideally the proposed nerve block needle trajectory should be routinely scanned with Color Flow Doppler. In addition, veins are collapsible and may be identified by applying and releasing pressure with help of ultrasound probe while scanning.

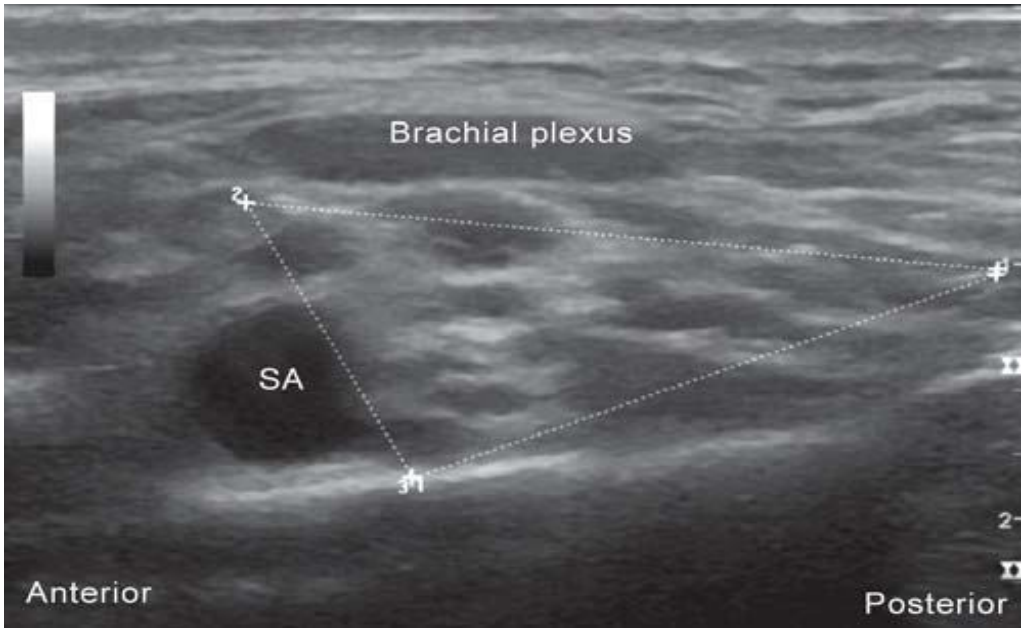


Fig. 5a: Supraclavicular brachial plexus arranged in triangular pattern. Plexus is seen as rounded to oblong hypoechoic structures surrounded by hyperechoic rim. Subclavian artery (SA) is seen as large rounded hypoechoic structure which is pulsatile in real time.

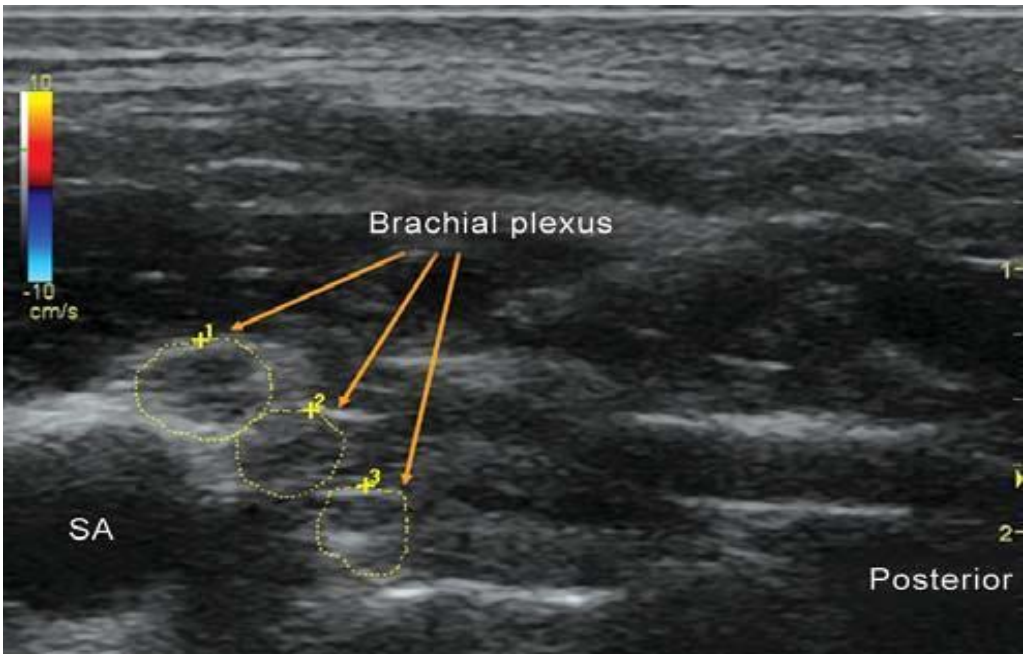


Fig. 5b: Supraclavicular brachial plexus arranged as vertical/obliquely arranged circles. Plexus is seen as hypoechoic round structure

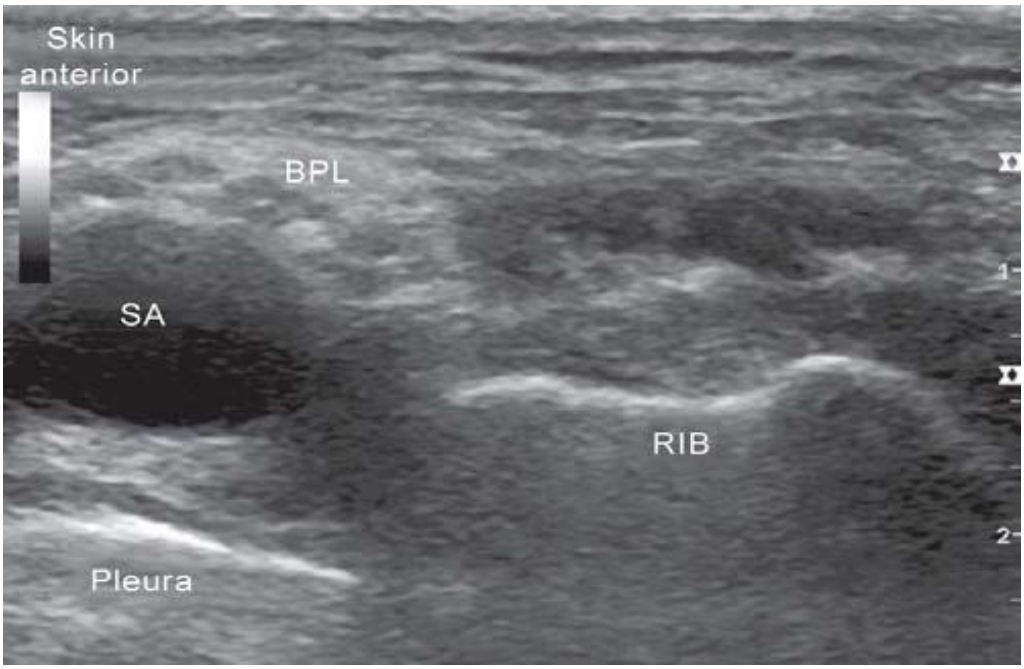


Fig. 5c: Showing pleura and rib. Rib is seen as linear hyperechoic area with acoustic shadowing, pleura is visualised as hyperechoic structure without acoustic shadow. SA, subclavian artery; BPL, brachial plexus.

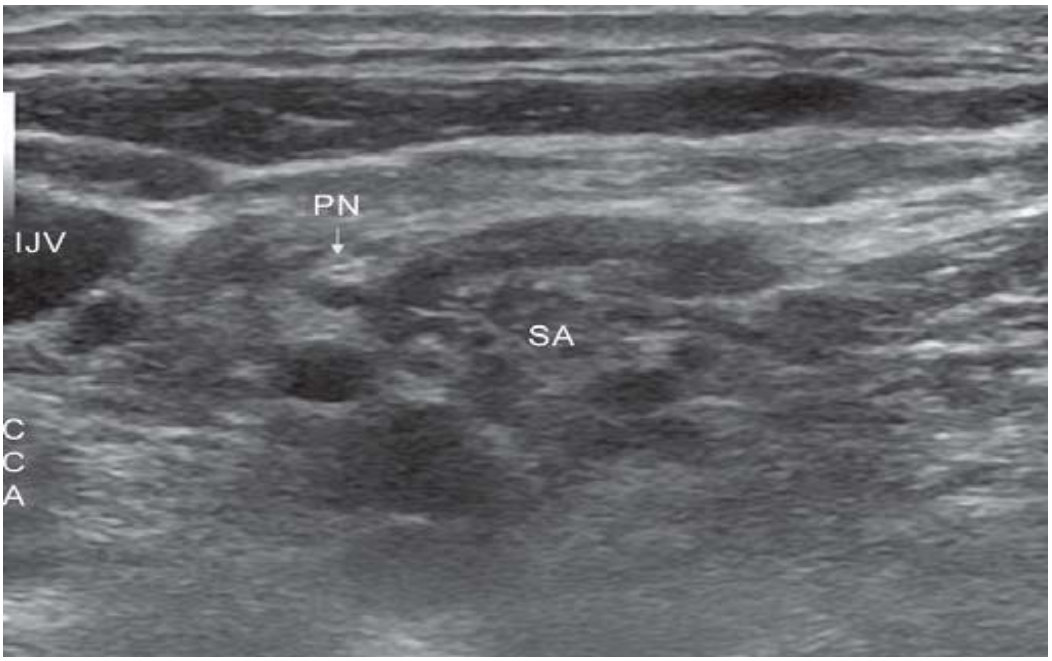


Fig. 5d: Showing phrenic nerve (PN) as hyperechoic structure on anterior surface of scalenus anterior (SA) muscle. IJV, internal jugular vein; CCA, common carotid artery

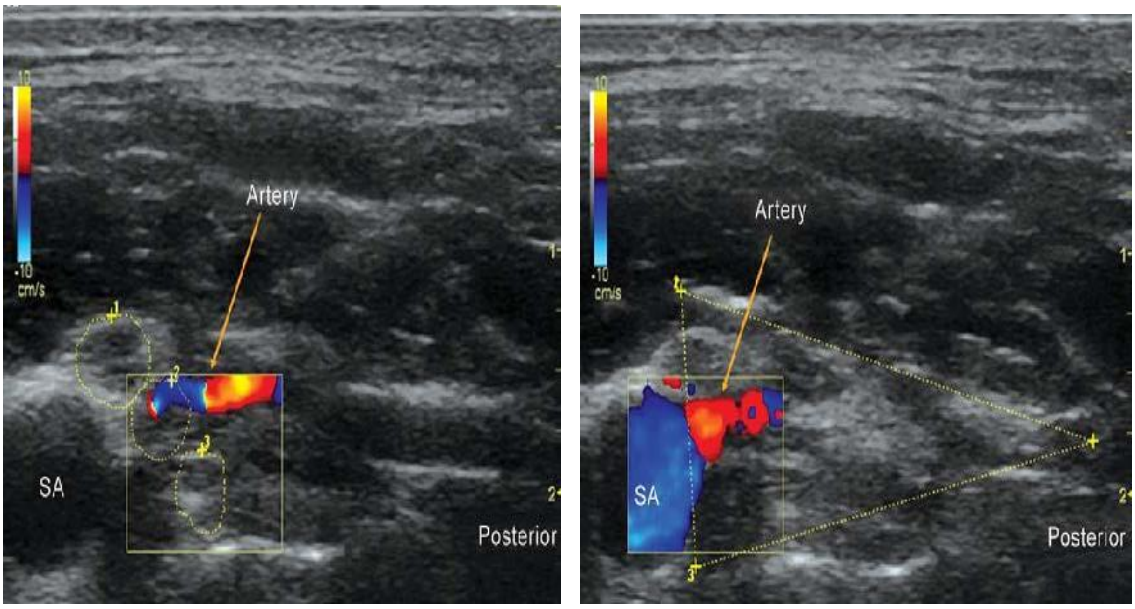


Fig 5e: Showing a branch of subclavian artery coursing through brachial plexus on colour doppler. SA, subclavian artery

INDICATIONS

Supraclavicular brachial plexus block produces rapid, reliable anaesthesia for surgical procedures of upper extremity.

Areas blocked are – Arm, forearm and hand except area over tip of shoulder (C3,C4) and inner aspect of upper arm (T2, intercostobrachial nerve).

CONTRAINDICATIONS

- General – patient refusal, allergy, disorder of hemostasis, preexistent neurologic deficit, respiratory failure, infection at site.
- Specific-particular stature(short neck, stiff neck)
- Associated disease – goiter.
- Radiotherapy sequel, past history of cervical node resection, contralateral recurrent laryngeal nerve palsy.

LOCAL ANESTHETIC MECHANISMS IN NERVE BLOCKADE

Impulse blockade by local anaesthetics may be summarized by the following chronology:

- Solutions of local anaesthetic are deposited near the nerve. Removal of free drug molecules away from this locus is a function of tissue binding, removal by the circulation, and local hydrolysis of amino-ester anaesthetics. The net result is penetration of the nerve sheath by the remaining free drug molecules.
- Local anaesthetic molecules then permeate the nerve's axon membranes and reside there and in the axoplasm. The speed and extent of these processes depend on a particular drug's pKa and on the lipophilicity of its base and cation species.

- Binding of local anesthetic to sites on voltage-gated Na^+ channels prevents opening of the channels by inhibiting the conformational changes that underlie channel activation. Local anesthetics bind in the channel's pore and also occlude the path of Na^+ ions.
- During onset or recovery from local anesthesia, impulse blockade is incomplete and partially blocked fibers are further inhibited by repetitive stimulation, which produces an additional use-dependent blinding to Na^+ channels.
- One local anesthetic binding site on the Na^+ channel may be sufficient to account for the drug's resting (tonic) and use-dependent (phasic) actions. Access to this site may potentially involve multiple pathways, but for clinical local anesthetics, the primary route is the hydrophobic approach from within the axon membrane.
- The clinically observed rates of onset and recovery from blockade are governed by the relatively slow diffusion of local anesthetic molecule into and out of the whole nerve, not by their much faster binding and dissociation from ion channels. A clinically effective block that may last for hours can be accomplished with local anesthetic drugs that dissociate from Na^+ channels in a few seconds.

COMPLICATIONS [38]

Vascular puncture :

Internal jugular vein may be punctured at skin wheal infiltration. Simple digital compression is required before continuing, the likelihood of arterial puncture implies not to pinpoint behind and too medial from mid clavicle. Best is to withdraw and redirect the needle when perceiving artery pulsation at the needle tip.

Pleural puncture :

The most significant complication of supraclavicular approach for blocking brachial plexus is development of pneumothorax. The incidence of pneumothorax is one percent with this technique and much higher in inexperienced hands. A pneumothorax must be suspected when there is dyspnea, cough or pleuritic chest pain but the diagnosis can be confirmed only by chest x-ray.

Phrenic nerve block :

Phrenic nerve block occurs in 40-60% of patient because of spread of local anaesthetic to the anterior surface of anterior scalene muscle. The effect is avoided if anaesthetic is deposited deep on the middle trunk on division or cord. This is rarely symptomatic. Radiographic confirmation may be obtained.

Recurrent laryngeal nerve block :

It causes transient dysphonia, occurs in 1% of case and only on the right side because recurrent laryngeal nerve loops around the subclavian artery on the right side and arch of aorta on the left.

Nerve damage or neuritis :

It results from the needle trauma or faulty positioning of anaesthetised arm preoperatively. Other remote causes include excessive tourniquet time, concentrated solution with vasoconstrictor and susceptible host tissue.[39]

Horner's syndrome :

It consists of ptosis, miosis, anhydrosis and enophthalmos. It usually follows stellate ganglion block. It is found in 10% of cases, after interscalene block.

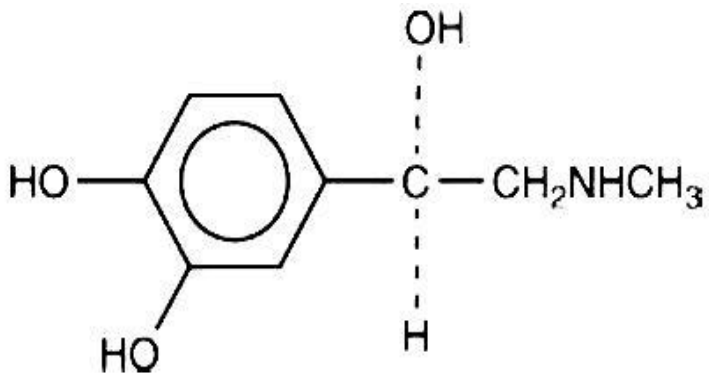
Toxic reaction to drug :

It is likely to occur if there is over dosage of drug or inadvertent intravascular injection is made, but can be avoided with proper negative aspiration test before drug injection.

PHARMACOLOGY OF BUPIVACAINE [40,41,42.]

INTRODUCTION

Bupivacaine is one of the homologous series synthesized in 1957 by A.F. Ekenstam to which Mepivacaine belongs. First report of its use was made in 1963 by Telivuo.[43] Bupivacaine is three to four times as potent as Lignocaine, and considerably longer lasting. It has the following structural formula:



Bupivacaine Hydrochloride is available in sterile isotonic solutions with and without epinephrine (as bitartrate) 1:2,00,000 for injection via local infiltration, peripheral nerve block, and caudal and lumbar epidural blocks. Solutions of Bupivacaine Hydrochloride may be autoclaved if they do not contain epinephrine. Solutions are clear and colorless. Bupivacaine is related chemically and pharmacologically to the aminoacyl local anaesthetics. It is a homologue of Mepivacaine and is chemically related to Lidocaine. All three of these anaesthetics contain an amide linkage between the aromatic nucleus and the amino, or Piperidine group. They differ in this respect from the procaine-type local anesthetics, which have an ester linkage.

Bupivacaine Hydrochloride Injection, USP is available in sterile, isotonic solutions containing Bupivacaine hydrochloride in water for injection with characteristics as follows:

TABLE-1: Bupivacaine Hydrochloride Injection, USP

(without epinephrine)

Concentration	Bupivacaine Hydrochloride mg/ml	Sodium Chloride mg/ml
0.25%	**2.5**	**8.6**
0.5%	**5**	**8.1**
0.75%	**7.5**	**7.6**

May contain sodium hydroxide or hydrochloric acid for pH adjustment. Multiple-dose vials contain methylparaben 1 mg/ml added as a preservative. Bupivacaine and Epinephrine Injection, USP is available in sterile, isotonic solutions containing Bupivacaine hydrochloride and epinephrine 1:200,000 with characteristics as follows :

TABLE-2: Bupivacaine and Epinephrine Injection, USP

Concentration (Bupivacaine HCL)	Bupivacaine Hydrochloride (mg/ml)	Epinephrine 1:2,00,000 (mcg/ml)
0.25%	**2.5**	**5**
0.5%	**5**	**5**
0.75%	**7.5**	**5**

Sodium metabisulfite 0.1 mg/ml added as antioxidant and edetate calcium disodium, anhydrous 0.1 mg/ml added as stabilizer. May contain sodium hydroxide or hydrochloric acid for pH adjustment. Multiple dose vials contain methylparaben 1 mg/ml added as preservative. Single-dose solutions contain no added bacteriostatic or anti-microbial agent and unused portions should be discarded after use.

Table-3: Physico-Chemical and Pharmacokinetic Properties

Molecular weight (KDa)	**288**
Pka	**8.1**
Lipid solubility	**30**
Protein binding (%)	**95**
Vd (L)	**73**
T1/2 (min)	**210**

PROPERTIES :

Bupivacaine hydrochloride, an amide is readily soluble in water and has good stability. The pH of plain solution is 6.0 to 6.7 and molecular weight is 324.9. It can be stored at room temperature. It is compatible with adrenaline and can be autoclaved more than twice. Commercially available Bupivacaine contains no preservative. The chemical name of Bupivacaine is (DL)-1-Butyl-2-(2,6-xylocarbonyl)-Piperidine.

MODE OF ACTION

It causes reversible blockade of sodium conduction probably by dual actions on cell membrane.

- They act directly on receptors within sodium channels.
- They produce nonspecific membrane expansion. Clinically the order of loss of nerve function is as follows :

1. Pain
2. Temperature
3. Touch
4. Proprioception
5. Skeletal muscle tone

PHARMACOLOGICAL EFFECTS

The effects produced by Bupivacaine may be:

1. **Local:** Nerve blockade and a direct effect on smooth muscle.
2. **Regional:** Loss of pain and temperature sensations, touch, motor power and vasomotor tone in the region supplied by the nerve blocked.
3. **Systemic :** The chief systemic effects are :
    a) **Cardiovascular system**: Gross overdose has been associated with ventricular tachycardia, fibrillation and cardiac arrest. There is good evidence, however that cardiac toxicity does not occur in subconvulsive doses or in absence of severe electrolyte disturbances or in absence of respiratory or metabolic acidosis. It causes vasodilatation in the area supplied by sympathetic nerves which are blocked.
    b) **Central nervous system :** It produces sedation and light headedness and sometimes anxiety and restlessness. With marked toxicity the patient may notice a numb tongue, circumoral pins and needles, twitching and visual disturbances. Severe toxicity proceeds to convulsions and coma with respiratory and cardiovascular depression.
    c) **Autonomic nervous system:** A weak blocking action on cholinergic and adrenergic receptors.
    d) **Neuromuscular junction:** It can block motor nerves if present in sufficient concentration.
    e) **Hypersensitivity:** It occurs more frequently in atopic patients in the forms of local edema, initially generalized urticaria or angioneurotic edema with or without lymphadenopathy. Dermatitis may be encountered as delayed reaction but anaphylaxis appear very rare.

PHARMACOKINETICS

Absorption

A dose of local anaesthetic is absorbed into the systemic circulation. Vascularity of tissue affect the space of absorption. So, it affects the toxicity.

Distribution

Bupivacaine has a great affinity for negatively charged protein receptor sites. At a plasma concentration of 1 mcg/ml the degree of protein binding is about 96.8% as opposed to 75% of Lignocaine. Thus it has high protein binding capacity.

Blood level

In animals 4 mcg/ml in plasma causes convulsions. The peak plasma concentration appears slowly and reaches highest between 5-30 minutes. After reaching this level it falls slowly, this explains to longer duration of action.

Placental transfer

As Bupivacaine is highly protein bound, it passes to fetus to a slower rate and is unlikely to cause fetal plasma concentration equal to that of maternal. Neonatal depression is not found with Bupivacaine.

Metabolism

It is rapidly catabolised like other local anaesthetics and chiefly metabolised in liver, metabolism involves N-dealkylation to pipecolyxylidine (PPX) which is then hydrolyzed. It has a fairly rapid rate of elimination from the blood because of faster tissue uptake and rapid rate of metabolism and so, there is hardly any accumulation of drug in the body even after prolonged administration and clinically found blood levels are much below the toxic dose.

Excretion

Demethylation of piperidene ring and coupling of glucuronic acid in the liver and is excreted through bile duct and kidney.

TABLE-4 : Routes and doses of administration

Local infiltration	**0.25%**
Peripheral nerve Block	**0.25% and 0.5%**
Retrobulbar block	**0.75%**
Sympathetic block	**0.25%**
Lumbar epidural	**0.25%, 0.5%, and 0.75%** **(0.75% not for obstetrical anesthesia)**
Caudal	**0.25% and 0.5%**
Epidural test dose	**0.5% with epinephrine 1:200,000**

Drug interactions

The administration of local anesthetic solutions containing epinephrine or norepinephrine to patients receiving monoamine oxidase inhibitors or tricyclic antidepressants may produce severe, prolonged hypertension. Concurrent use of these agents should generally be avoided. In situations when concurrent therapy is necessary, careful patient monitoring is essential. Concurrent administration of vasopressor drugs and of ergot-type oxytocic drugs may cause severe, persistent hypertension or cerebrovascular accidents. Phenothiazines and butyrophenones may reduce or reverse the pressor effect of epinephrine.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Long-term studies in animals of most local anesthetics including Bupivacaine to evaluate the carcinogenic potential have not been conducted. Mutagenic potential or the effect on fertility has not been determined. There is no evidence from human data that Bupivacaine Hydrochloride may be carcinogenic or mutagenic or that it impairs fertility.

Adverse Reactions/ Toxicity

Toxicity depends on the amount of free drug in plasma; this relates to three factors:

1. Dose given.

2. Rate of injection (the effective dose given).

3. Site of injection (the greater the blood supply to the area injected the greater the systemic absorption). Sites of absorption from greatest to least:

Interpleural > Intercostal > Pudendal > Caudal > Epidural > Brachial plexus > Infiltration.

Reactions to Bupivacaine Hydrochloride are characteristic of those associated with other amide-type local anaesthetics. The most commonly encountered acute adverse experiences which demand immediate counter-measures are related to the central nervous system and the cardiovascular system. These adverse experiences are generally dose related and due to high plasma levels which may result from over dosage, rapid absorption from the injection site, diminished tolerance, or from unintentional intravascular injection of the local anesthetic solution.

In addition to systemic dose-related toxicity, unintentional subarachnoid injection of drug during the intended performance of caudal or lumbar epidural block or nerve blocks near the vertebral column (especially in the head and neck region) may result in under ventilation or apnea (“Total or High Spinal”). Also, hypotension due to loss of sympathetic tone and respiratory paralysis or under ventilation due to cephalad extension of the motor level of anesthesia may occur. This may lead to secondary cardiac arrest if untreated. Patients over 65 years, particularly those with hypertension, may be at increased risk for experiencing the hypotensive effects of Bupivacaine Hydrochloride. Factors influencing plasma protein binding, such as

acidosis, systemic diseases which alter protein production, or competition of other drugs for protein binding sites, may diminish individual tolerance.

Central Nervous System Reactions

The principal effect of Bupivacaine is reversible neuronal blockade which leads to characteristic biphasic effect on the CNS.

These are characterized by excitation and/or depression. Restlessness, anxiety, dizziness, tinnitus, blurred vision, or tremors may occur, possibly proceeding to convulsions.

However, excitement may be transient or absent, with depression being the first manifestation of an adverse reaction. This may quickly be followed by drowsiness merging into unconsciousness and respiratory arrest.

Other central nervous system effects may be nausea, vomiting, chills, and constriction of the pupils. The incidence of convulsions associated with the use of local anesthetics varies with the procedure used and the total dose administered.

Cardiovascular System Reactions

Bupivacaine depresses the rapid phase of depolarization (Vmax) in purkinje fibers and ventricular musculature greater than Lignocaine. It is a “Fast in, Slow out” local anaesthetic .It also decreases the rate of recovery from dependant block than that of Lignocaine . This leads to incomplete restoration of Vmax between action potentials at high rate , in contrast to complete recovery by Lignocaine .This explains the arrythmogenic potential of Bupivacaine . High levels of Bupivacaine prolongs conduction through various parts of heart and extremely high concentrations will depress spontaneous pacemaker activity ,resulting in bradycardia and arrest .

CC/CNS dose ratio is 3.7 ± 0.5.

CC/CNS blood level ratio; 1.6 ± 1.7. [44]

High doses or unintentional intravascular injection may lead to high plasma levels and related depression of the myocardium, decreased cardiac output, heart block, hypotension, bradycardia, ventricular arrhythmias, including ventricular tachycardia and ventricular fibrillation, and cardiac arrest.

Respiratory system

Respiratory depression may be caused if excessive plasma level is reached which in turn results in depression of medullary respiratory centre . Respiratory depression may also be caused by paralysis of respiratory muscles as may occur in high spinal or total spinal anaesthesia.

Autonomic nervous system

Myelinated preganglionic beta fibers have a faster conduction time and are more sensitive to the action of local anaesthetics including Bupivacaine. When used for conduction blockade Bupivacaine produces higher incidence of sensory blockade than motor fibers.

Allergic reaction

Allergic-type reactions are rare and may occur as a result of sensitivity to the local anesthetic or to other formulation ingredients, such as the antimicrobial preservative methylparaben contained in multiple-dose vials or sulfites in epinephrine containing solutions.

These reactions are characterized by signs such as urticaria, pruritus, erythema, angioneurotic edema (including laryngeal edema), tachycardia, sneezing, nausea, vomiting, dizziness, syncope, excessive sweating, elevated temperature, and possibly, anaphylactoid-like symptomatology (including severe hypotension).

Cross sensitivity among members of the amide-type local anesthetic group has been reported. The usefulness of screening for sensitivity has not been definitely established.

PHARMACOLOGY OF ROPIVACAINE

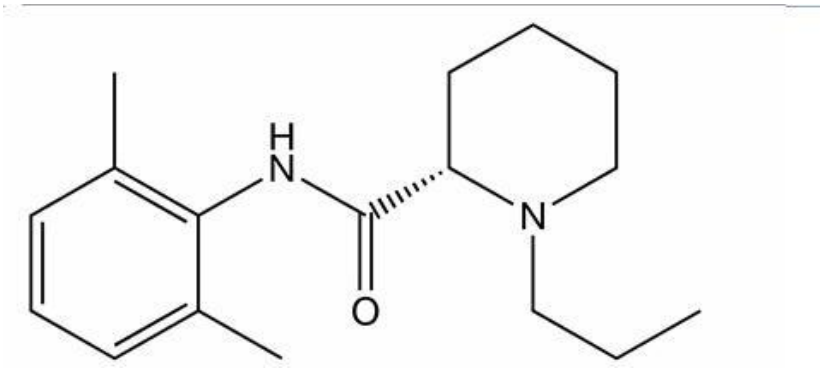
INTRODUCTION

One of the most important properties of a long-acting local anaesthetics is to reversibly inhibit the nerve impulses, thus causing a prolonged sensory or motor blockade appropriate for anaesthesia in different types of surgeries.[45]

The acute pain relief obtained at lower doses in postoperative and labour patients due to sensory blockade is sometimes marred by accompanying motor blockade, which serves no purpose and is quite undesirable.

Ropivacaine is a long-acting local anaesthetic that is structurally related to Bupivacaine. It is a pure S(-) enantiomer, unlike Bupivacaine, which is a racemate, developed for the purpose of reducing potential toxicity and improving relative sensory and motor block profiles.[46]

STEREOSPECIFICITY AND STRUCTURE



C17H26N2O

Enantiomers exist in two different spatial configurations, and are present in equal amounts in a racemic solution. They are optically active and can be differentiated by their effects on the rotation of the plane of a polarised light into dextrorotatory [clockwise rotation (R+)] or levorotatory [counterclockwise rotation (S-)] stereo-isomers.

The physicochemical properties of the two enantiomeric molecules are identical, but the two enantiomers can have substantially different behaviours in their affinity for either the site of action or the sites involved in the generation of side effects. R(+) and S(-) enantiomers of local anaesthetics have been demonstrated to have different affinity for different ion channels of sodium, potassium, and calcium; this results in a significant reduction in central nervous system (CNS) and cardiac toxicity (cardiotoxicity) of the S(-)enantiomer as compared with the R(+)enantiomer.[46]

The technological advancements have made it possible to develop Ropivacaine as an optically pure S(-) enantiomer from the parent chiral molecule propivacaine. It belongs to the group of local anaesthetics, the pipecoloxylidides and has a propyl group on the piperidine nitrogen atom compared to Bupivacaine, which has a butyl Group.[47]

Preparations

- Single dose container in concentration

  0.20% 0.50%

  0.75% 1.00%

- 5mg/ml in 20 ml vial
- 5 mg/ml in 10 ml ampoule
- 10mg/ml in 10ml ampoule

- Ropivacaine is available as isotonic solution that contains the enantiomerically pure drug substance, sodium chloride for isotonicity and water for Injection. Sodium hydroxide and/or hydrochloric acid may be used for pH adjustment.
- At 25°C Ropivacaine HCl has a solubility of 53.8 mg/ml in water, specific gravity of 1.002-1.005 , a distribution ratio between n-octanol and phosphate buffer at pH 7.4 of 14:1 and a pKa of 8.07 in 0.1 M KCl solution. The pKa of Ropivacaine is approximately the same as Bupivacaine (8.1) and is similar to that of Mepivacaine (7.7).

TABLE -5 : Physico-Chemical and Pharmacokinetic Properties

Molecular weight (KDa)	**274**
Pka	**8.1**
Lipid solubility	**2.8**
Protein binding (%)	**94**
Vd (L)	**59**
T1/2 (min)	**111**

MECHANISM OF ACTION

Ropivacaine causes reversible inhibition of sodium ion influx, and thereby blocks impulse conduction in nerve fibers. This action is potentiated by dose-dependent inhibition of potassium channels.[48] Ropivacaine is less lipophilic than Bupivacaine and is less likely to penetrate large myelinated motor fibers. Therefore, it has selective action on the pain-transmitting Aα and C nerves rather than Aβ fibers, which are involved in motor function.

PHARMACODYNAMICS

Central Nervous System and Cardiovascular effects

Ropivacaine is less lipophilic than Bupivacaine and that, together with its stereoselective properties,[49] contributes to Ropivacaine having a significantly higher threshold for cardiotoxicity and CNS toxicity than Bupivacaine in healthy volunteers[50,51,52]

The lower lipophilicity of Ropivacaine versus Bupivacaine correlated with the lesser cardiodepressant effects of both Ropivacaine isomers than of the Bupivacaine isomers.[49]

The Central Nervous System effects occurs earlier than cardiotoxic symptoms during an intravenous (IV) infusion of local anaesthetic (10 mg/min of Ropivacaine or Bupivacaine). Significant changes in cardiac function involving the contractility, conduction time and QRS width occurred and the increase in a QRS width was found to be significantly smaller with Ropivacaine than with Bupivacaine.[53]

Other effects

Ropivacaine has been shown to inhibit platelet aggregation in plasma at concentrations of 3.75 mg/ml (0.375%), which correspond to those that could occur in the epidural space during infusion.[54] Like other anaesthetics, Ropivacaine has antibacterial activity in vitro, inhibiting the growth of Staphylococcus Aureus, Escherichia coli, and Pseudomonas aeruginosa.[55,56]

PHARMACOKINETICS

Absorption and distribution

The plasma concentration of Ropivacaine depends on the total dose administered and the route of administration, as well as the hemodynamic and circulatory condition of the patient and vascularity of the administration site. [57]

When Ropivacaine is administered intravenously , its pharmacokinetics are linear and dose proportional. The absorption of Ropivacaine from the epidural space is complete and biphasic. The mean half-life of the initial phase is approximately 14 minutes, followed by a slower phase with a mean absorption $t_{1/2}$ of approximately 4.2 hours. Ropivacaine is bound to plasma proteins to an extent of 94%, mainly to α1-acid glycoprotein.

Ropivacaine rapidly crosses the placenta resulting in near complete equilibrium of the free fraction of Ropivacaine in the maternal and fetal circulation.[58] However, the total plasma concentration of Ropivacaine remains lower in the fetal circulation than in the maternal circulation, reflecting the binding of Ropivacaine to α1-acid glycoprotein, which is more concentrated in maternal than in fetal plasma.

Metabolism and excretion

Ropivacaine is metabolised extensively in the liver, predominantly by aromatic hydroxylation to 3′-hydroxy-ropivacaine by cytochrome P450 (CYP) 1A2 and N-dealkylation to 2′,6′-pipecoloxylidide by CYP3A4.[59,60] The kidney is the main excretory organ for Ropivacaine, accounting for 86% of the excretion of the drug in urine after a single intravenous dose administration. It has a mean ± SD terminal half-life of 1.8±0.7 hours and 4.2 ± 1.0 hours after intravenous and epidural administration, respectively.

Relative potency

A strict correlation exists between the lipid solubility of the local anesthetic and its potency and toxicity. According to minimum local anesthetic concentration (MLAC) studies, which are based on effective analgesia in 50% of patients, Ropivacaine has similar potency to Bupivacaine at higher doses (eg, doses required for peripheral nerve blocks for surgical anaesthesia), Ropivacaine is less potent than Bupivacaine and Levobupivacaine at lower doses, such as those used for epidural or intrathecal analgesia. Providing anaesthesia or analgesia for the majority of patients is more clinically relevant than the MLAC and at higher doses used in clinical practice, this potency difference is not always evident.

Tolerability

Reactions to Ropivacaine are characteristic of those associated with other amide-type local anaesthetics.

a) **In Adults:** Ropivacaine is generally well tolerated regardless of the route of administration. The cardiovascular events are related to toxicity due to sudden IV injection or massive absorption from peripheral nerve blocks.

b) **In Children:** [61] Ropivacaine is generally well tolerated in paediatric patients aged from 1 month to 15 years regardless of the route of administration. The overall incidence of adverse events associated with Ropivacaine appears to be low, with nausea and vomiting occurring most frequently.

c) **In exposed fetuses and neonates:** Ropivacaine is generally well tolerated in the fetus or neonate following the use of regional anaesthesia in women undergoing caesarean section or during labour. The most common fetal or neonatal adverse events with Ropivacaine are fetal bradycardia, neonatal jaundice and unspecified neonatal complications.

CARDIOTOXICITY AND CENTRAL NERVOUS SYSTEM TOXICITY IN COMPARISON TO BUPIVACAINE

The incidence of cardiotoxicity and central nervous system (CNS) toxicity as a result of inadvertent intravascular injection of Ropivacaine appears to be low.[62]

The convulsive local anaesthetic doses of Bupivacaine and Ropivacaine were studied in different animal models; Bupivacaine has a 1.5- to 2.5-fold lower convulsive threshold when compared to Ropivacaine. On the basis of animal and volunteer studies, it can be concluded that Ropivacaine seems to be less neurotoxic and cardiotoxic than Bupivacaine.

CC/CNS Dose ratio of Ropivacaine : 4.8 ± 0.4

DRUG INTERACTIONS

Ropivacaine should be used with caution in patients receiving other local anaesthetics or agents structurally related to amide-type local anaesthetics, since the toxic effects of these drugs are additive.

Cytochrome P4501A2 metabolises Ropivacaine to 3-hydroxy Ropivacaine, the major metabolite. Thus, strong inhibitors of cytochrome P4501A2, such as fluvoxamine, given concomitantly during administration of Ropivacaine, can interact with Ropivacaine and thus lead to increased Ropivacaine plasma levels. Caution should be exercised when co-administering CYP1A2 inhibitors. Possible interactions with drugs known to be metabolized by CYP1A2 via competitive inhibition such as theophylline and imipramine may also occur.

TABLE – 6: Routes and doses of administration

The dosage recommendations for Ropivacaine in various indications and procedures are summarized in table 6.

Indication in adults	Concentration (%)	Volume	Dose
Surgical anaesthesia			
Lumbar epidural	0.75	15-20 ml	113-150 mg
Caesarean section			
Lumbar epidural	0.75	15-25 ml	113-188 mg
(Other surgery)	1	15-20 ml	150-200 mg
Thoracic (Single block for postoperative pain relief)	0.75	5-15 ml	38-113 mg

Indication in adults	Concentration (%)	Volume	Dose
Intrathecal administration	0.5	3-4 ml	15-20 mg
Peripheral nerve block[*]	0.75	10-40 ml	75-300 mg
Field block[†]	0.75	1-30 ml	7.5-225 mg
Postoperative pain			
Lumbar epidural (Continuous infusion)	0.2	6-10 ml/h	12-20 mg/h
Thoracic epidural (Continuous infusion)	0.2	6-14 ml/h	12-28 mg/h
Peripheral nerve block (Continuous infusion)	0.2	5-10 ml/h	10-20 mg/h
Field block[†]	0.2	1-100 ml	2-200 mg
Intra-articular injection	0.75	20 ml	150 mg

Indication in adults	Concentration (%)	Volume	Dose
Labour pain (Lumbar epidural)			
Bolus	0.2	10-20 ml	20-40 mg
Intermittent top-ups	0.2	10-15 ml[‡]	20-30 mg
Continuous infusion	0.2	6-14 ml/h	12-28 mg/h
In children			
Caudal epidural block (Below T12) [§]	0.2	1 ml/kg	2 mg/kg
Peripheral nerve block (Eg, ilioinguinal block)	0.5	0.6 ml/kg	3 mg/kg

[*]Major nerve block brachial plexus or sciatic nerve block

[†]Minor nerve block or infiltration

[‡]Minimum interval 30 minutes

[§]For bodyweight up to 25 kg.

CLINICAL APPLICATIONS

Numerous clinical trials have evaluated the efficacy of Ropivacaine for surgical anaesthesia, for labour pain and postoperative pain in adults and children. The drug has been compared primarily with Bupivacaine or Levobupivacaine. In the recent years, the use of Ropivacaine in the management of chronic pain has also been evaluated in various modalities.

Surgical anaesthesia

Clinical trials indicate that Ropivacaine is an effective regional anaesthetic when administered via several routes.

Epidural administration

Epidural Ropivacaine, administered primarily in the lumbar region, has an effect of anaesthetic for a number of surgical procedures. A majority of studies on epidural Ropivacaine are in Caesarean section and although the drug has been investigated as an anaesthetic agent for other abdominal or gynecological procedures, orthopedic and vascular surgery, the major use of epidural Ropivacaine in the latter procedures is for postoperative pain relief.

a) **Caesarean section:** [63] Clinical trials of epidural anaesthesia for elective Caesarean section indicate that Ropivacaine (0.75% or 0.5%) provides a clinically similar onset of sensory and motor block to that of Bupivacaine 0.5%.

b) **Hip or lower limb surgery:** In patients undergoing lumbar epidural anaesthesia for lower limb surgery, Ropivacaine provided a similar anaesthetic profile (with regard to onset of analgesia or anaesthesia and onset of motor block) to those of Levobupivacaine.[64]

INTRATHECAL ADMINISTRATION

Single doses of 2-4 ml of 0.5%-1% solutions of Ropivacaine have been shown to be less potent than Bupivacaine when administered intrathecally and is generally administered at a higher dose than Bupivacaine. Hyperbaric solutions of Ropivacaine have been compared to isobaric solution of the drug for various procedures and generally resulted in a faster onset and recovery from the blocks. Although hyperbaric solutions of Ropivacaine appear to provide a more predictable block, the spread and duration of the block varies markedly. Hyperbaric Ropivacaine solutions are not commercially available. The co-administration of opioids reduces the total dose of local anaesthetic required for anaesthesia and significantly prolongs the duration of complete and effective analgesia without prolonging the duration of motor block.[65]

It is also suggested that on a milligram for milligram basis, the potency of Ropivacaine relative to Bupivacaine is two-thirds with regard to sensory block and half with regard to motor block.[66]

PERIPHERAL NERVE BLOCKS

Peripheral nerve block is employed for anaesthesia for orthopedic surgery, and the onset and spread of local anaesthetic is influenced by the site of injection.[67]

The long-acting sensory and motor block provided by Ropivacaine 0.5% or 0.75% for axillary, interscalene and subclavian perivascular brachial plexus block for hand or arm surgery compared favorably with Bupivacaine 0.5% or Levobupivacaine 0.5%(30-45 ml bolus dose) with a similar quality of regional anaesthesia.[67,68,22,24,69.]

In lower limb surgeries where sciatic or combined femoral and sciatic block are given for knee, ankle, or foot procedures, Ropivacaine 0.75% (25 ml) had a significantly faster onset of sensory and motor block than 25 ml Bupivacaine 0.5%.

Although Ropivacaine had a significantly shorter duration of sensory block, the duration of motor block remained similar with both agents.[70]

MANAGEMENT OF POSTOPERATIVE PAIN

Lower doses of local anaesthetics are generally required for postoperative pain relief than for anaesthesia.

Epidural administration

Ropivacaine is administered epidurally (via lumbar or thoracic route) for postoperative pain following abdominal (upper or lower), gynecological, orthopedic and other surgeries.

Pain relief with epidural Ropivacaine 0.2% was not as effective as Bupivacaine 0.2% in patients who had undergone knee arthroplasty.[71]

Nerve blocks :

a) **Following upper limb surgery:** [72] There appears similar pain relief with Ropivacaine and Bupivacaine, although hand strength returns more quickly and there was less paraesthesia of the fingers in patients receiving Ropivacaine than in those receiving Bupivacaine.

b) **Following lower limb surgery:** [70] Patients who received combined femoral and sciatic nerve block with Ropivacaine to facilitate foot/ankle surgery had similar or better postoperative pain relief and a longer duration of analgesia than recipients of Mepivacaine.

MANAGEMENT OF LABOUR PAIN

Epidurally administered Ropivacaine is effective in providing relief from labour pain. It is recommended to administer 10-20 ml bolus of Ropivacaine 0.2% with intermittent 20-30 mg top up injections or a continuous epidural infusion of Ropivacaine 0.2% (6-10 ml/hr) for labour analgesia. The analgesic efficacy of Ropivacaine is similar to or slightly less than Bupivacaine.

The addition of narcotics like fentanyl 2 μg/ml to Ropivacaine 0.1% solution significantly reduces local anaesthetic doses.[73] Addition of adjuvants like clonidine also significantly increases the duration of action of Ropivacaine.

Intrathecally administered Ropivacaine as a part of combined spinal epidural technique produces rapid and effective labour pain relief with less incidence of motor Block.[74]

Adverse Reactions

Adverse drug reactions are rare when it is administered correctly. Most adverse drug reactions relate to administration technique (resulting in systemic exposure) or pharmacological effects of anesthesia, however allergic reactions can rarely occur. These adverse experiences are generally dose-related and due to high plasma levels that may result from over dosage, rapid absorption from the injection site, diminished tolerance or from unintentional intravascular injection of the Ropivacaine solution.

In addition to systemic dose-related toxicity, unintentional subarachnoid injection of drug during the intended performance of lumbar epidural block or nerve blocks near the vertebral column (especially in the head and neck region) may result in underventilation or apnea ("Total or High spinal").

Also, hypotension due to loss of sympathetic tone and respiratory paralysis or under ventilation due to cephalic extension of the motor level of anesthesia may occur. This may lead to secondary cardiac arrest if untreated.

Factors influencing plasma protein binding such as acidosis, systemic diseases that alter protein production or competition with other drugs for protein binding sites, may diminish tolerance. Systemic exposure to excessive quantities of Ropivacaine mainly result in central nervous system (CNS) and cardiovascular effects – CNS effects usually occur at lower blood plasma concentrations and additional cardiovascular effects present at higher concentrations, though cardiovascular collapse may also occur with low concentrations.

Central Nervous System effects

Include CNS excitation (nervousness, tingling around the mouth, tinnitus, tremor, dizziness, blurred vision, seizures followed by depression (drowsiness, loss of consciousness, respiratory depression and apnea).

Cardiovascular effects

Include hypotension, bradycardia, arrhythmias, and cardiac arrest – some of which may be due to hypoxemia secondary to respiratory depression**.**

Gastrointestinal effects

Nausea ,vomiting .

Genitourinary

Urinary retention (5%), oliguria, urinary tract infection.

Hypersensitivity

Allergic-type reactions, such as urticaria, pruritus, erythema, angioneuritic edema, including laryngeal edema, tachycardia, sneezing, nausea, vomiting, dizziness, syncope, excessive sweating, elevated temperature, and anaphylactoid symptomatology, including severe hypotension are seen rarely .

Respiratory

Dyspnea (1% to 5%); rhinitis (1%); respiratory arrest, respiratory paralysis, underventilation or apnea.

Contraindications

Ropivacaine is contraindicated for intravenous regional anaesthesia (IVRA). However, new data suggested Ropivacaine (1.2-1.8 mg/kg in 40ml) can be used, because it has less cardiovascular and central nervous system toxicity than Bupivacaine.

Treatment of overdose

As for Bupivacaine, Celepid , an intravenous lipid emulsion, can be effective in treating severe cardiotoxicity secondary to local anaesthetic overdose in humans in a process called lipid rescue.[75] Intravenous intralipid (20%) appears to be effective at minimizing adverse cardiovascular outcomes . It is believed that the lipid acts as a bank for local anaesthetic – the drug has more affinity for the lipid than for cardiac tissue, as the amount of Buipivicaine/ Ropivacaine bound up to cardiac tissue is reduced, normal contractile function results. Intralipid is inexpensive and has a long shelf life.

Initial bolus of 100ml (1.5ml/kg over 1 minute) followed by 0.25 ml/kg over 20 minutes. Repeat boluses can be administered subsequently. 100ml at 5 minute intervals repeated twice and then 400ml administered over 10 minutes. CPR should be continued until the circulation has been re-established. If all of this fails – cardiopulmonary bypass may be instituted until the local anaesthetic has been metabolized.

MATERIALS AND METHODS

SOURCE OF DATA:

The present study entitled "A Comparative study of inj.Bupivacaine 0.5% and inj.Ropivacaine 0.5% for supraclavicular Brachial plexus block" for upper limb orthopedic surgery was carried out in the Department of Anaesthesiology, B.L.D.E. University's Shri B.M Patil Medical College, Hospital and Research Centre, Vijayapur.

METHOD OF COLLECTION OF DATA:

Study period – One and half year from October 2013 to June 2015 .

Study design – Randomized Comparative study.

Sample size – In a study, it was found that the mean ± S.D of onset times for analgesia in Ropivacaine and Bupivacaine was 15±15 and 31±56 min respectively.[16]

Considering the average of S.D. of onset time of both the drugs 35.5 minutes and at 0.05 alfa error, 0.20 beta error the total sample size was 78 using the following statistical formula :

$$n = \frac{(Z_\alpha + Z_\beta)^2 \times 2 \times SD^2}{d^2}$$

where, Z_α = Z value at α level 95%

Z_β = Z value at β level 90%

SD = Standard deviation

d = differences between parameters

A total of 78 patients were randomly allocated into group B and group R. Hence 39 cases were included in each of the groups.

STATISTICAL ANALYSIS :

Data was analyzed using

- Diagrams
- Mean ± S.D
- Student 't' test or suitable non parametric test
- Chi-Square test.

INCLUSION CRITERIA:

- patients of either sex aged between 18 to 60 years.
- patients with ASA grade I and II.
- weight 50 to 80 kgs.
- Patients scheduled for elective upper limb surgeries.

EXCLUSION CRITERIA:

- Patients with known hypersensitivity to study local anaesthetics.
- Patients refusal.
- Patients with coagulopathy or patients on anticoagulation therapy.
- Patients with documented neuromuscular disorders.
- Patients with severe hepatic, renal, respiratory or cardiac diseases.
- Cutaneous infection at the site of block.
- ASA Grade III and IV

PRE-ANAESTHETIC EVALUATION :

During preoperative visit, patients detailed history, general physical examination and systemic examination were carried out. Basic demographic characters like age, sex, weight were recorded.

INVESTIGATIONS REQUIRED :

- Hb%, TC, DC, BT, CT.
- Urine analysis.
- RBS, Blood urea, serum creatinine, and serum electrolytes.
- Chest x-ray, ECG if required.
- HBsAg, HIV (Universal precautions)

PRELIMINARIES :

- Patients were premedicated with Tab.Alprazolam 0.5 mg night before surgery.
- Written informed consent was taken from the patient.
- NBM for 6 hours was confirmed.
- Intravenous access secured with 20 guaze i.v canula on the contralateral upper limb under aseptic precautions.

RANDOMIZATION :

Each patient was randomly allocated to one of the two groups of 39 patients each by computer generated randomization table.

Group B – Bupivacaine group received 30 ml Bupivacaine 0.5% (5 mg/ml)

Group R – Ropivacaine group received 30 ml Ropivacaine 0.5% (5 mg/ml)

EQUIPMENTS :

SonoSite M Turbo Ultrasound Machine

a) **For the procedure :**

A portable tray covered with sterile towels containing :

- Sterile syringes – one 20ml and one 10ml.
- Bowl containing povidine iodine and spirit.
- Sponge holding forceps.
- Towels and towel clips.
- Sterile gauze pieces.
- Study drug.
- Disposable hypodermic needle 22 gauge.

b) **For emergency resuscitation :**

- Anaesthesia machine, emergency oxygen source (E type cylinders), pipeline oxygen supply, working laryngoscopes, appropriate size endotracheal tubes and connectors.
- Working suction apparatus with suction catheter.
- Oropharyngeal airways
- Intravenous fluids
- Drugs : Thiopentone, diazepam, succinylcholine, hydrocortisone, atropine, adrenaline, aminophylline, mephenteramine, calcium gluconate and sodium bicarbonate.

c) **Monitors :**

- Pulse oximeter.
- ECG
- NIBP monitor.

PROCEDURE: After the patient was taken on the operating table, intravenous access secured in the upper limb opposite to that undergoing surgery with a large bore i.v. cannula. Standard multi parameter monitor, ECG, pulse oximeter, non invasive blood pressure were connected and monitored in all the patients.

POSITIONING: Patient was placed in supine position with the head turned away from the side to be blocked. Arm to be anaesthetized adducted and extended towards the ipsilateral knee as far as possible which will depress the clavicle slightly and allow better access to the structures of the anterolateral neck. Also, a slight elevation of the head of the bed is often more comfortable for the patient and allows for better drainage and less prominence of the neck veins.

IMAGE ACQUISITION: With the patient in proper position the supraclavicular area is aseptically prepared and draped and a linear 38-mm, high frequency 10-15 MHz transducer is placed firmly over the supraclavicular fossa in the coronal oblique plane to obtain the best possible transverse view of the subclavian artery and brachial plexus. Nerves in the supraclavicular region appear hypo-echoic and are round or oval. The brachial plexus is located lateral and superficial to the pulsatile subclavian artery and superficial to the first rib. The subclavian artery is identified first, subclavian vein lies more medially. The first rib is identified as a hyper-echoic structure lying deep to the vessels, and giving a bony shadow. The brachial plexus is consistently found lateral and superficial to the subclavian artery and above the first rib.

NEEDLE PLACEMENT: For the in plane approach (lateral to medial) a 5 cm 22G insulated block needle is inserted under sterile conditions on the outer (lateral) end of the ultrasound transducer (5-12 or 6-13 MHz) after skin local anaesthetic infiltration. The brachial plexus is identified as a compact group of

nerves, sometimes compared to a ‘bunch of grapes’ , located over the first rib, lateral and superficial to the subclavain artery. The rib and pleura are identified before needle insertions. The needle is advanced along the long axis of the transducer in the same plane as the ultrasound beam.

This way the needle shaft and tip can be visualized in real time as the needle is advanced towards the target nerves. The identity of the nerves may be confirmed by electrical stimulation if desired. Useful stimulation endpoints for surgery proximal to elbow are biceps and triceps twitches; hand muscle twitches are more appropriate for surgery distal to the elbow. After negative aspiration for blood, 30 ml of respective local anaesthetic drug was injected depending on whether patient is allotted to either of group B or R so as to cause hydro dissection of the planes around the plexus. Local anaesthetic spread is observed during injection and the needle repositioned to ensure distribution around all the nerve trunks and divisions within the plexus sheath. No sign of local anaesthetic spread may indicate intravascular injection and so care must be taken when this occurs to re-identify the needle tip before further local anaesthetic injection.

In plane (medial to lateral) approach may also be used based on user comfort. Inj. Bupivacaine 0.25% 5ml will be given to block intercostobrachial nerve (T_2) to avoid tourniquet pain.

Onset of sensory blockade, onset of motor blockade, duration of sensory blockade, duration of motor blockade and any adverse effects were noted.

Fig.6: Drugs used for study.

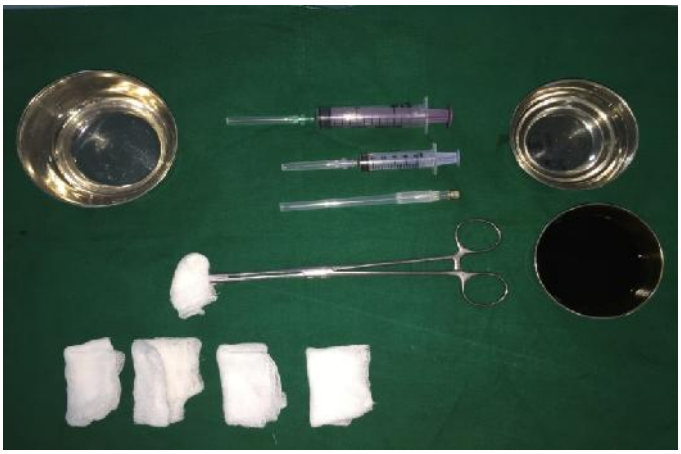


Fig.7: Sterile tray containing drug and equipments.

Fig.8: SonoSite M-Turbo Ultrasound Machine

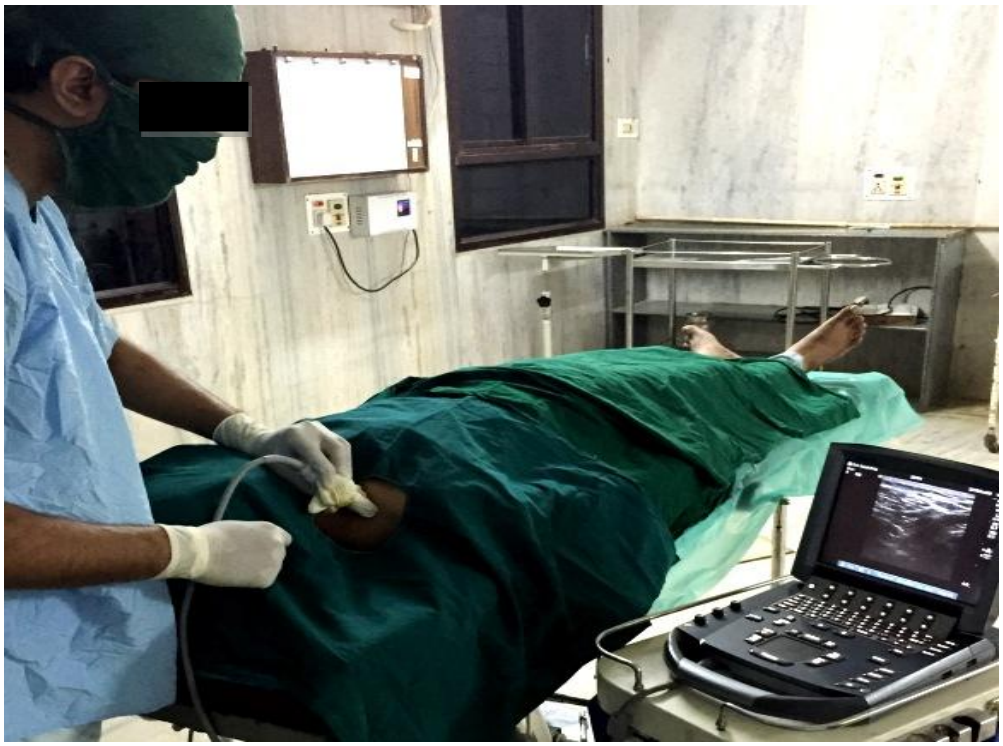


Fig. 9: Ultrasound probe placed in oblique coronal plane.

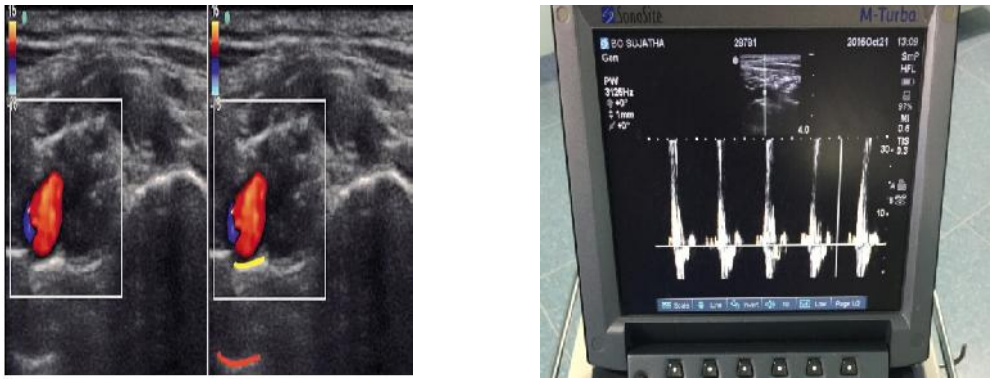


Fig.10: Doppler for identification of vessels. Indicated for both initial identification of subclavian vessels and aberrant vessels traversing the plexus before choosing the needle trajectory.

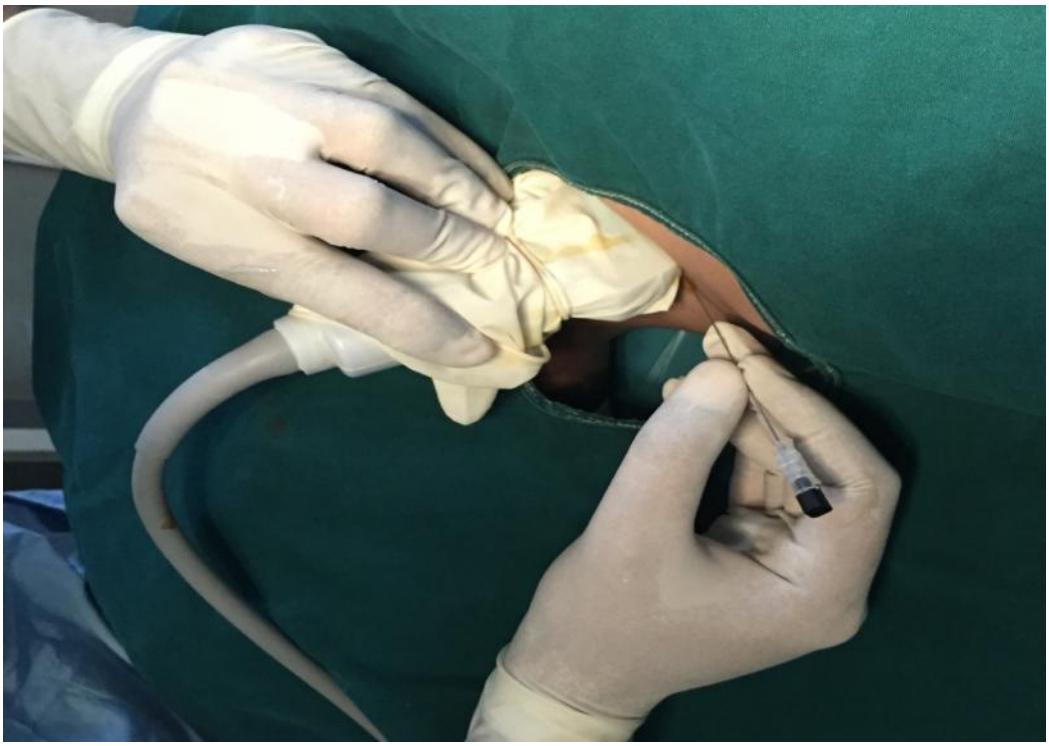


Fig 11:Transducer position and needle insertion .

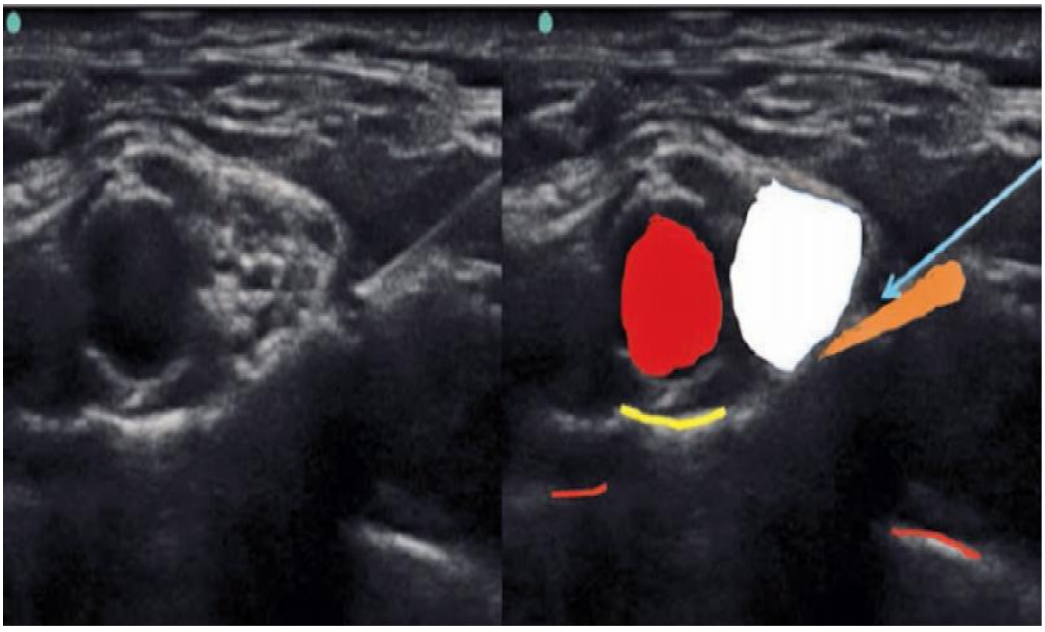


Fig.12: Scout scan of supraclavicular fossa and needle insertion in plane. Red area: subclavian artery, white area: brachial plexus, yellow line: periostium of first rib, orange line: periostium of clavicle, red line: pleura, blue arrow: needle.

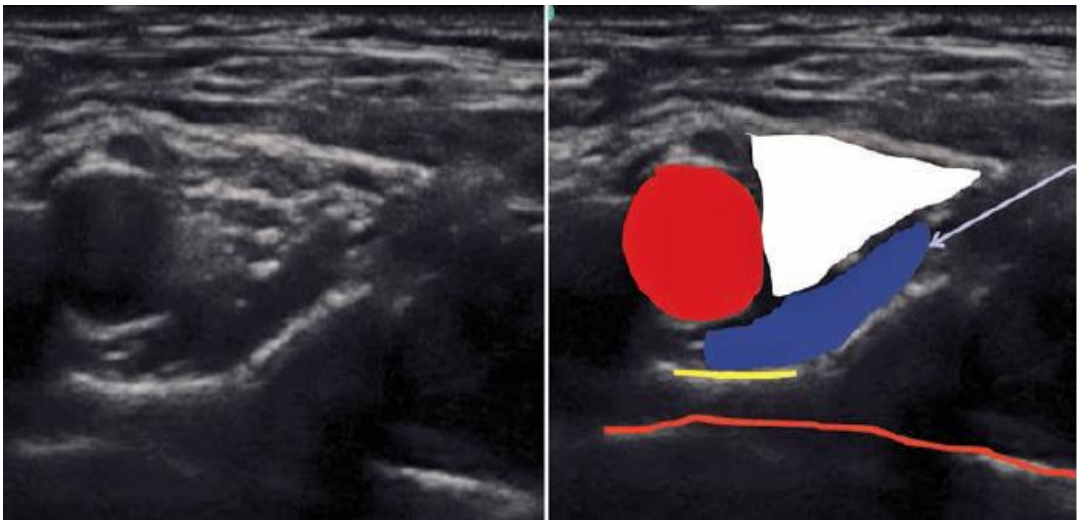


Fig.13: Spread of local anaesthetic solution deep to the plexus. Red area: subclavian artery, white area: brachial plexus, yellow line: periostium of first rib, red line: pleura, blue arrow: needle, navy area: local anaesthetic.

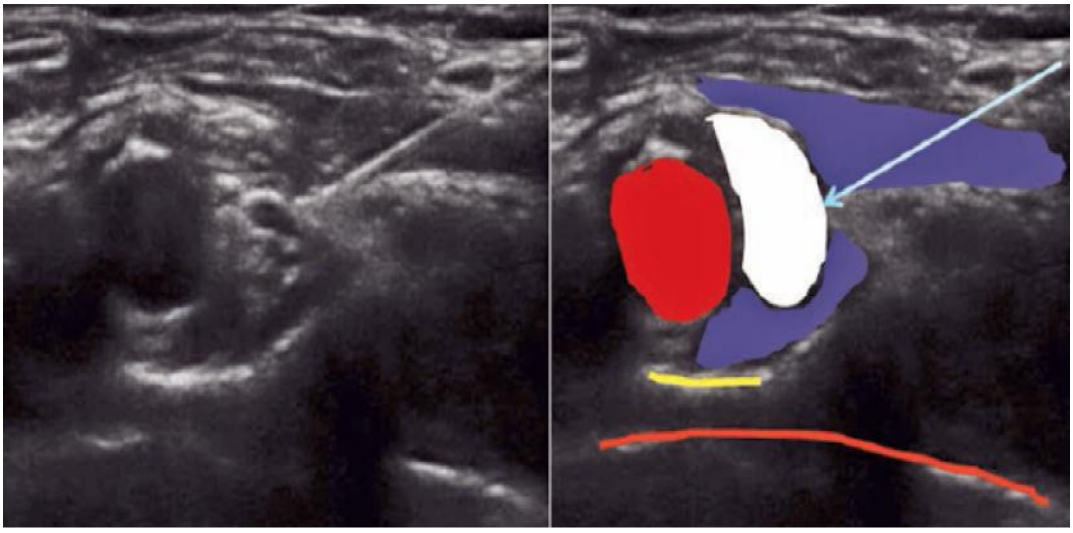


Fig.14: Spread of local anaesthetic solution superficial to the plexus. Red area: subclavian artery, white area: brachial plexus, yellow line: periostium of first rib, red line: pleura, blue arrow: needle, navy area: local anaesthetic

ASSESSMENT OF SENSORY BLOCK

Sensory block was assessed by pin prick with 23 guaze hypodermic needle in skin dermatomes C_4-T_2 once in every minute for initial 30 minutes and then after every 30 minutes till patient regained normal sensations and graded according to Visual analogue scale (VAS) as

0-No Pain

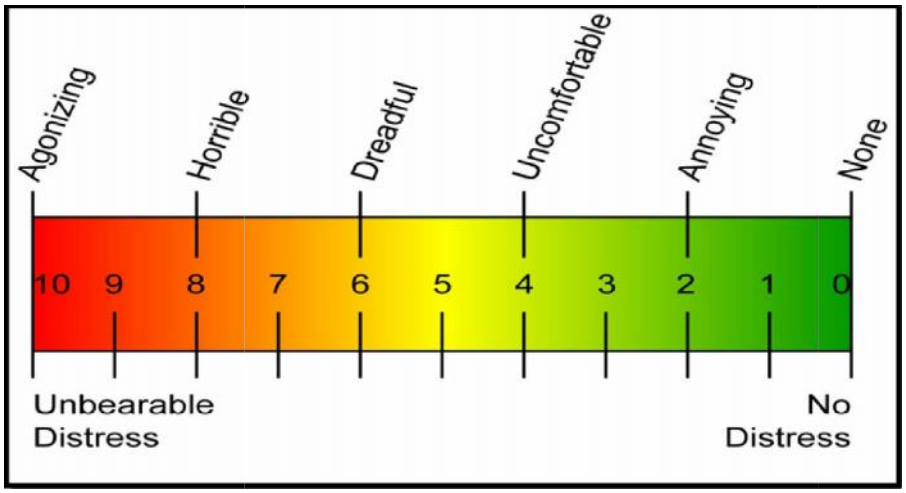
2-Annoying (Mild pain)

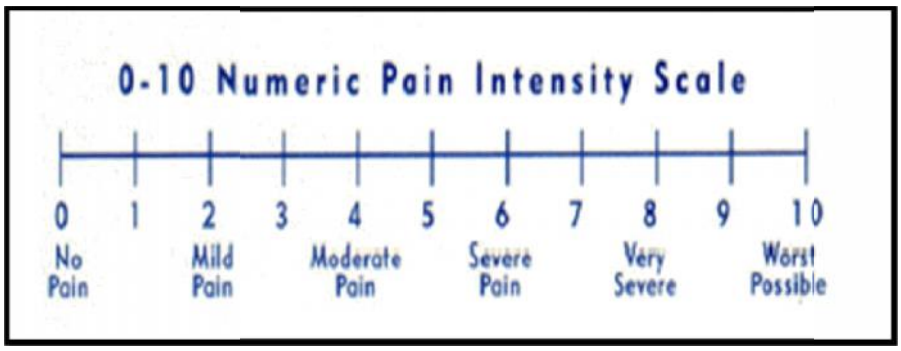
4-Uncomfortable (Moderate pain)

6-Dreadful (Severe pain)

8-Horrible (Very severe pain)

10-Agonizing (Worst possible pain)





ASSESSMENT OF MOTOR BLOCK

Quality of motor block was assessed at the same intervals and graded according to Modified Lovett's Scoring as

Grade 6- Normal

Grade 5 –Slightly reduced muscular force

Grade 4 – Pronounced reduction.

Grade 3 – Slightly impaired mobility.

Grade 2 – Pronounced mobility impairment.

Grade 1 – Almost complete paralysis.

Grade 0– Complete paralysis.

The effect on the following parameters were observed :

Onset of motor blockade- time taken from the completion of injection of study drug till the patient develops motor blockade.(Lovett"s Grade 1)

Onset of sensory blockade- time taken from the completion of injection of study drug till the patient does not feel the pin prick.(Visual analogue scale score -0)

Duration of motor blockade- time taken from the onset of motor blockade till complete recovery of motor power. (Lovett's grade 6)

Duration of sensory blockade – time taken from the onset of sensory blockade till the patient feels pin prick. (Visual analogue scale of 2)

Patients were watched for bradycardia, convulsions, restlessness, disorientation, drowsiness, nausea, vomiting & any other complications.

All the values were expressed as Mean ± Standard deviation, statistical comparison was performed by student's t-test & chi-square test.

A two tailed P value of >0.05 was considered to be statistically not significant, < 0.05 as statistically significant, < 0.01 as statistically highly significant.

RESULTS

The present study was conducted on 78 consenting patients aged between 18-60 years. Group B received 30 ml of 0.5% Bupivacaine and Group R received 30 ml of 0.5% Ropivacaine for supraclavicular brachial plexus block.

DEMOGRAPHIC DATA

TABLE 7: PERCENT DISTRIBUTION OF GENDER

Gender	N	Percent
Male	50	64.1
Female	28	35.9
Total	78	100

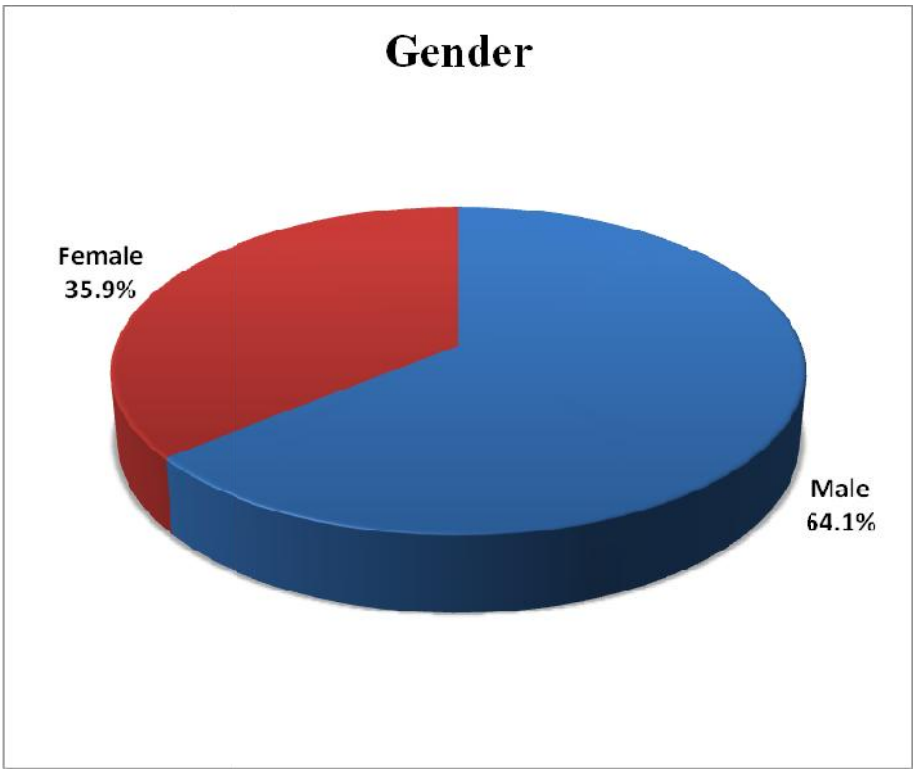


FIGURE - 15: PERCENT DISTRIBUTION OF GENDER

TABLE 8 : PERCENT DISTRIBUTION OF AGE

Age (Yrs)	N	Percent
18-30	24	30.8
31-40	25	32.0
41-50	22	28.2
51-60	7	9.0
Total	78	100

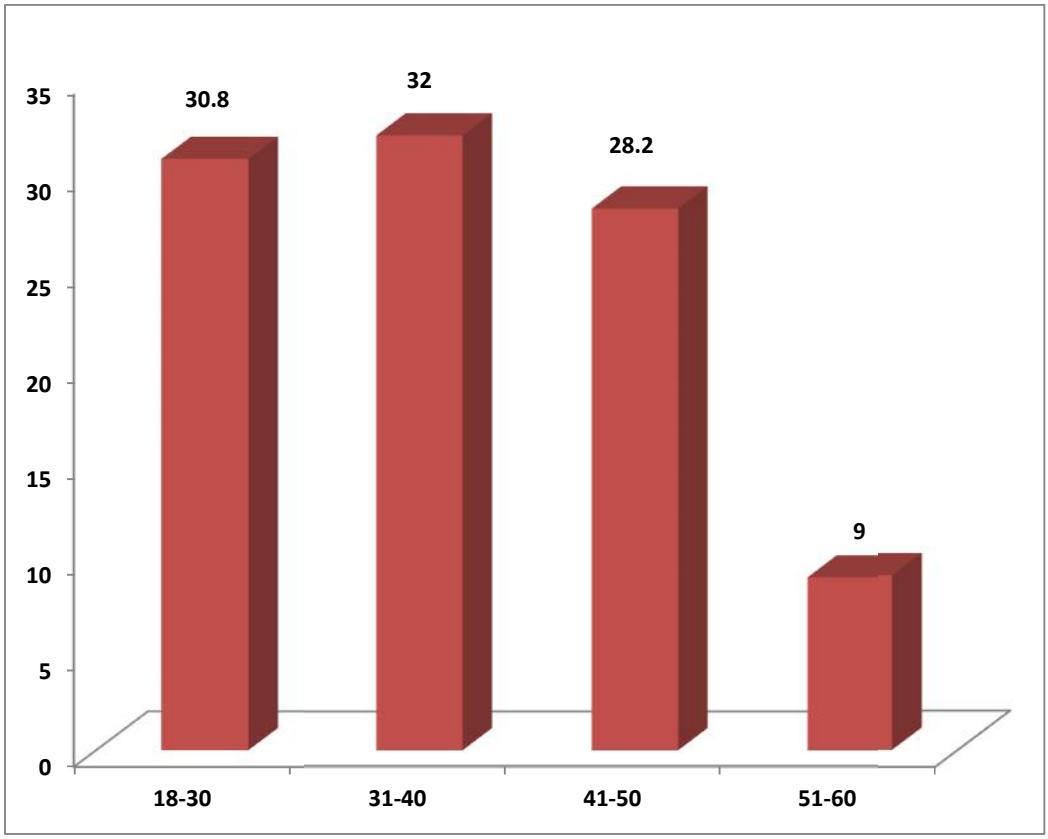


FIGURE - 16: PERCENT DISTRIBBUTION OF AGE

TABLE 9: PERCENT DISTRIBUTION OF WEIGHT

Weight(Kg)	N	Percent
50-60	13	16.7
61-70	34	43.6
71-80	31	39.7
Total	78	100

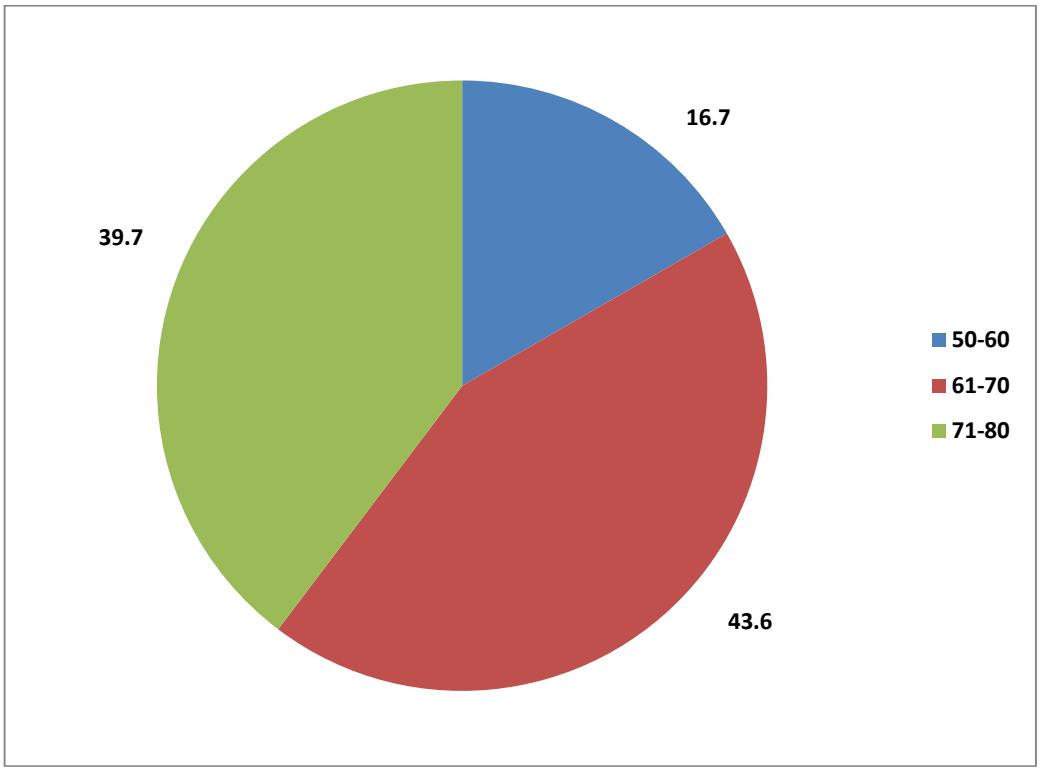


FIGURE - 17 : PERCENT DISTRIBUTION OF WEIGHT

TABLE 10: AGE DISTRIBUTION BETWEEN STUDY GROUPS

Age(Yrs)	BUPIVACAINE (N=39)		ROPIVACAINE (N=39)	
	N	Percent	N	Percent
18-30	12	30.8	12	30.8
31-40	14	35.9	11	28.2
41-50	7	17.9	15	38.4
51-60	6	15.4	1	2.6
Total	39	100.0	39	100.0
Mean±SD	36.2±10.6		36.9±9.1	

Samples are age matched with P value 0.758

As shown in table 10 , both the groups ,Group B (Bupivacaine) and Group R (Ropivacaine) are age matched .

In age group of 18-30 years there are 12 patients in both groups .

In the age group of 31-40 years, there are 14 patients in group B and 11 patients in group R .

In the age group of 41-50 years, there are 7 patients in group B and 15 patients in group R.

In the age group of 51-60 years, there are 6 patients in group B and 1 patient in group R.

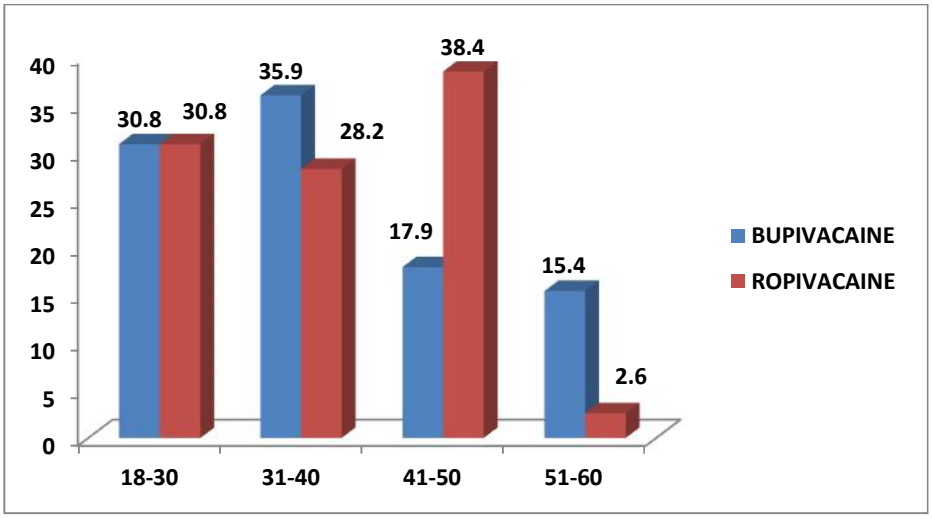


FIGURE - 18: AGE DISTRIBUTION BETWEEN STUDY GROUPS

TABLE 11: GENDER DISTRIBUTION BETWEEN STUDY GROUPS

Gender	BUPIVACAINE (N=39)		ROPIVACAINE (N=39)		Total	p value
	N	Percent	N	Percent		
Female	13	46.4	15	53.6	28	0.637
Male	26	52.0	24	48.0	50	

As shown in table 11, both the groups, Group B (Bupivacaine group) and Group R (Ropivacaine group) are gender matched. There are 26 males in group B and 24 males in Group R , 13 females in group B and 15 females in group R.

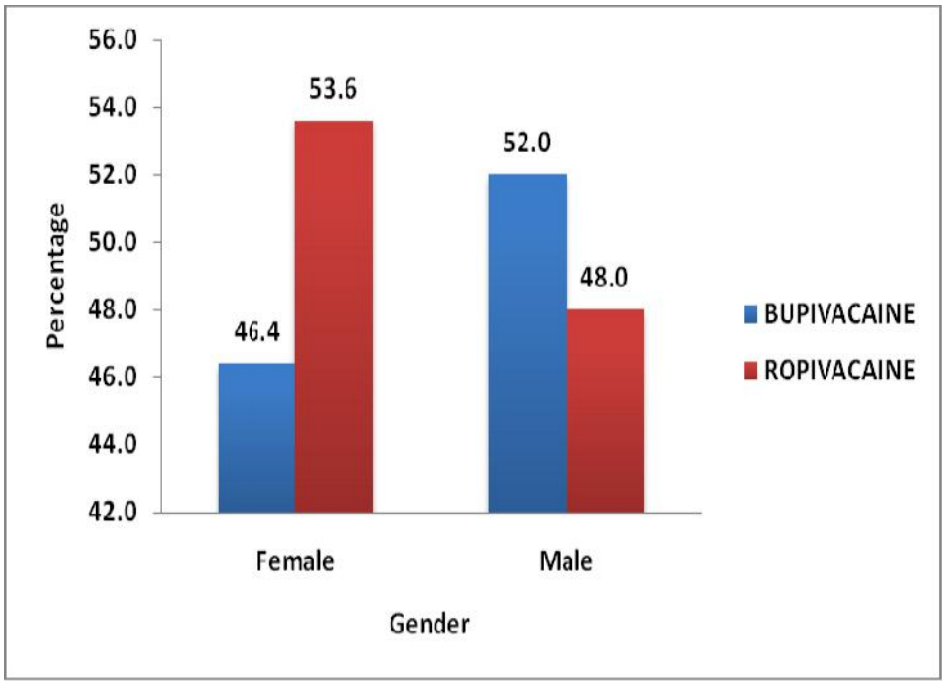


FIGURE - 19 : GENDER DISTRIBUTION BETWEEN STUDY GROUPS

TABLE 12: ASSOCIATION OF WEIGHT (Kg) BETWEEN STUDY GROUPS

Weight	BUPIVACAINE (N=39)		ROPIVACAINE (N=39)		Total	p value
	N	Percent	N	Percent		
50-60	8	61.5	5	38.5	13	0.55
61-70	15	44.1	19	55.9	34	
71-80	16	51.6	15	48.4	31	

As shown in table 12, both the groups, group B and group R are matched with respect to weight of the patients.

There are 8 patients in group B and 5 patients in group R with weight between 50kg- 60 kg.

There are 15 patients in group B and 19 patients in group R with weight between 61 kg- 70 kg.

There are 16 patients in group B and 15 patients in group R with weight between 71 kg- 80 kg.

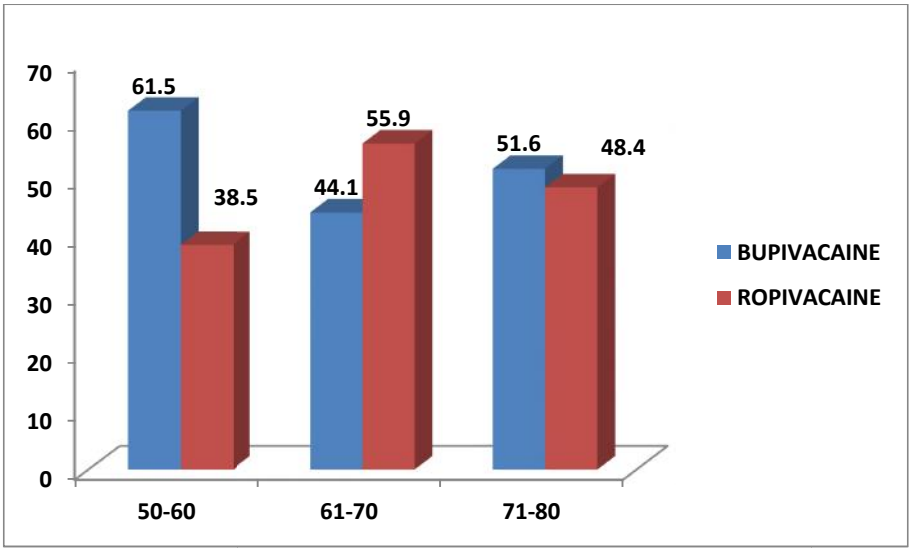


FIGURE – 20 : ASSOCIATION OF WEIGHT(Kg) BETWEEN STUDY GROUPS

TABLE 13: MEAN COMPARISON OF HEART RATE(per min) BETWEEN STUDY GROUPS.

Heart rate (per min)	BUPIVACAINE (N=39)		ROPIVACAINE (N=39)		p value
	Mean	SD	Mean	SD	
0 min	89.3	6.5	87.5	6.8	0.891
5 min	88.9	6.8	87.2	5.3	0.81
15 min	88.5	6.7	86.9	6.0	0.545
30 min	88.1	6.0	86.6	6.7	0.187
60 min	87.7	6.2	86.3	7.5	0.756
2 hrs	87.3	5.7	86.0	6.3	0.737
3 hrs	86.9	6.7	85.7	6.0	0.285
6 hrs	86.5	6.2	85.4	6.0	0.604
12 hrs	86.1	6.6	85.1	7.1	0.052

As shown in table 13, Heart rate variation between the groups, group B and group R, was not statistically significant (p>0.05) .

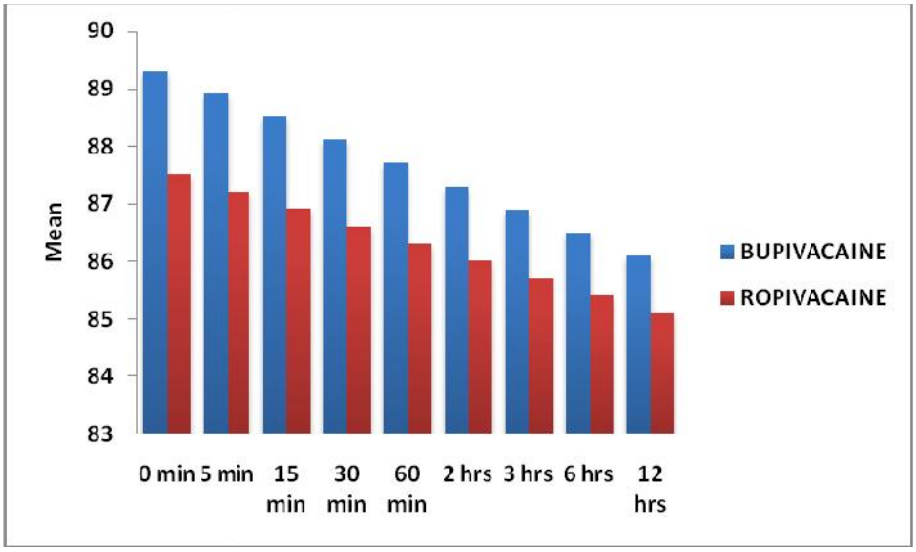


FIGURE – 21: MEAN COMPARISON OF HEART RATE (per min) BETWEEN STUDY GROUPS

TABLE 14: MEAN COMPARISON OF SYSTOLIC BLOOD PRESSURE (mm of hg) BETWEEN STUDY GROUPS

Systolic BP (mm of Hg)	BUPIVACAINE (N=39)		ROPIVACAINE (N=39)		p value
	Mean	SD	Mean	SD	
0 min	124.1	1.9	123.2	1.8	0.855
5 min	122.1	1.4	122.3	1.4	0.463
15 min	119.2	1.7	119.3	1.8	0.846
30 min	116.6	1.0	115.5	1.1	0.607
60 min	115.6	1.0	114.5	1.1	0.607
2 hrs	113.6	1.0	113.5	1.1	0.607
3 hrs	112.6	1.0	112.5	1.1	0.607
6 hrs	111.6	1.0	111.5	1.1	0.607
12 hrs	109.6	1.0	109.5	1.1	0.607

As shown in table 14, systolic blood pressure (mm of Hg) variation between the groups , group B and group R, was not statistically significant (p>0.05).

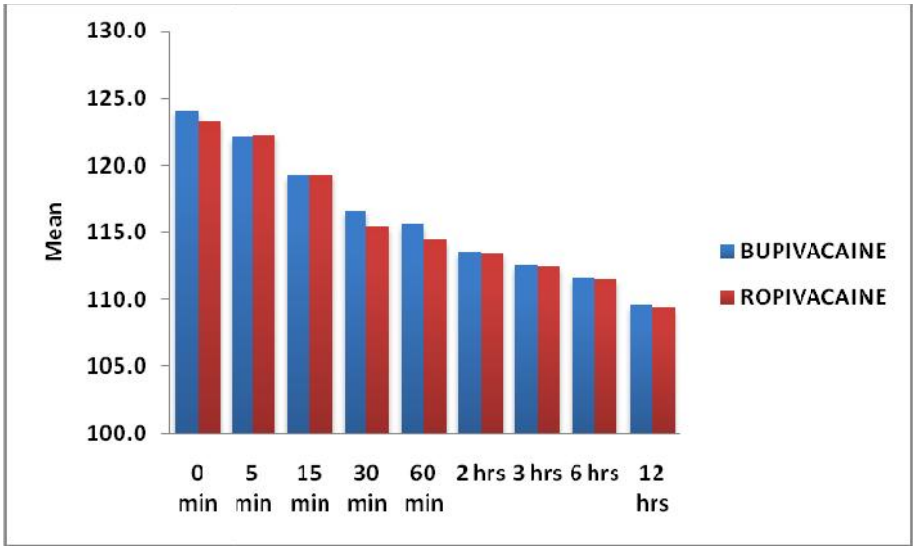


FIGURE - 22: MEAN COMPARISION OF SYSTOLIC BLOOD PRESSURE (mm of Hg) BETWEEN STUDY GROUPS

TABLE 15: MEAN COMPARISON OF DIASTOLIC BLOOD PRESSURE (mm of hg) BETWEEN STUDY GROUPS

Diastolic BP (mm of Hg)	BUPIVACAINE (N=39)		ROPIVACAINE (N=39)		p value
	Mean	SD	Mean	SD	
0 min	78.1	2.5	77.9	2.5	0.686
5 min	77.1	2.5	76.9	2.4	0.716
15 min	75.3	2.1	75.1	2.0	0.666
30 min	74.3	2.1	74.1	2.0	0.666
60 min	71.8	2.1	72.3	2.0	0.274
2 hrs	71.6	1.3	71.2	1.2	0.199
3 hrs	71.4	1.2	71.5	1.2	0.78
6 hrs	71.5	1.0	71.4	1.2	0.919
12 hrs	71.5	1.1	71.2	1.2	0.388

As shown in table 15, Diastolic blood pressure (mm of Hg) variation between the groups , Group B and Group R, was not statistically significant (p>0.05).

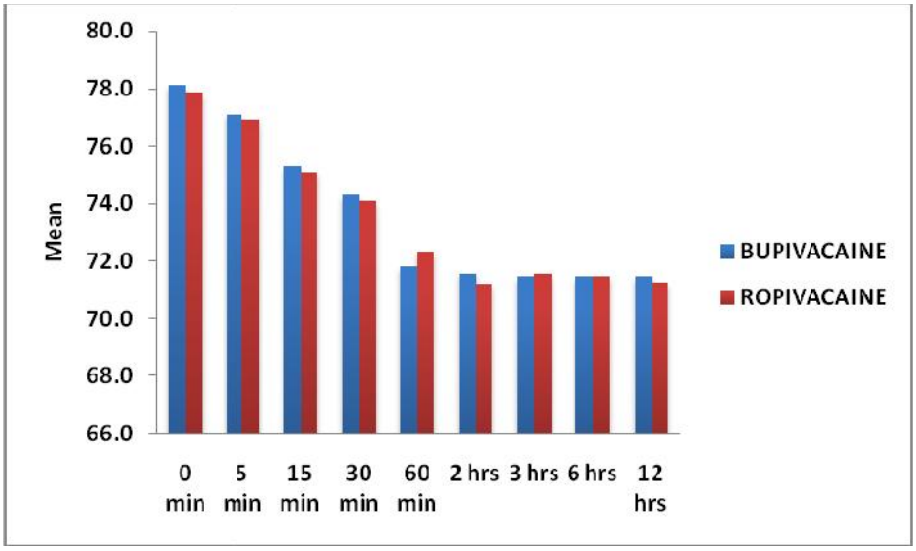


FIGURE - 23 : MEAN COMPARISION OF DIASTOLIC BLOOD PRESSURE (mm of Hg) BETWEEN STUDY GROUPS.

TABLE 16: COMPARISON OF DURATION OF SURGERY BETWEEN STUDY GROUPS

Parameters	BUPIVACAINE (N=39)		ROPIVACAINE (N=39)		p value
	Mean	SD	Mean	SD	
Duration of Surgery (Min)	126.4	33.0	116.2	36.5	0.196

As seen in table 16, There was no statistically significant difference found between the two groups with respect to the duration of surgery (p>0.05).

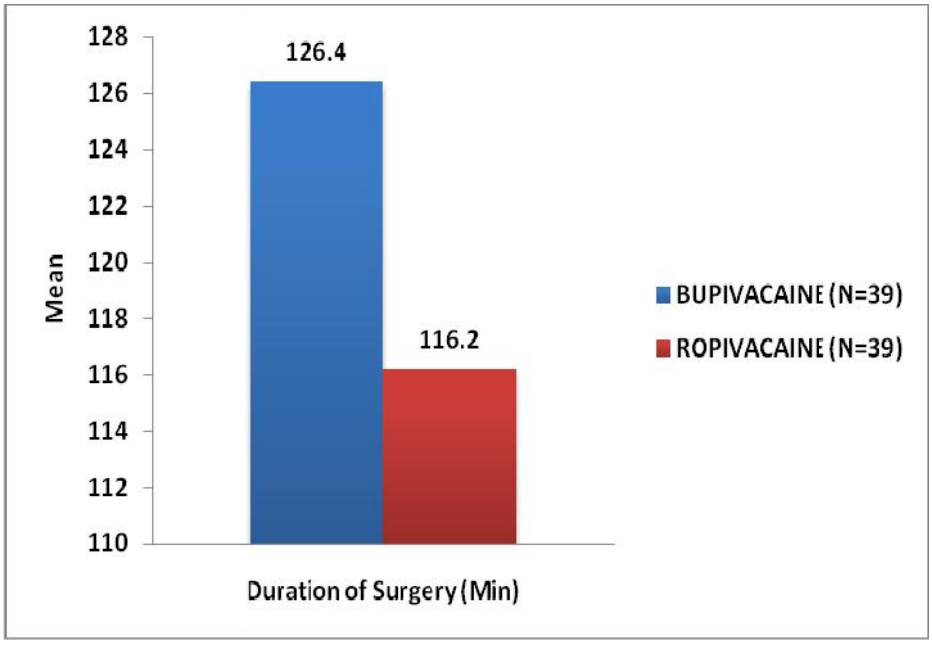


FIGURE - 24: COMPARISON OF DURATION OF SURGERY BETWEEN STUDY GROUPS

TABLE 17: COMPARISON OF GROUP B AND GROUP R ON THE BASIS OF ONSET TIME OF SENSORY AND MOTOR BLOCKADE.

Parameters	BUPIVACAINE (N=39)		ROPIVACAINE (N=39)		p value
	Mean	SD	Mean	SD	
Sensory Onset Time (Min)	16.6	3.2	19.9	4.0	0.0001
Motor Onset Time (Min)	21.4	2.6	25.9	2.4	0.000

In Group B , the mean onset time of sensory blockade and motor blockade was 16.6 ± 3.2 min and 21.4 ± 2.6 min respectively when compared to Group R having onset time of sensory blockade and motor blockade of 19.9 ± 4.0 min and 25.9 ± 2.4 min respectively.

Comparison of Mean Onset Time between the groups

Onset time of sensory blockade and motor blockade was earlier in Group B when compared with Group R. The p value was < 0.001 which was statistically very highly significant .

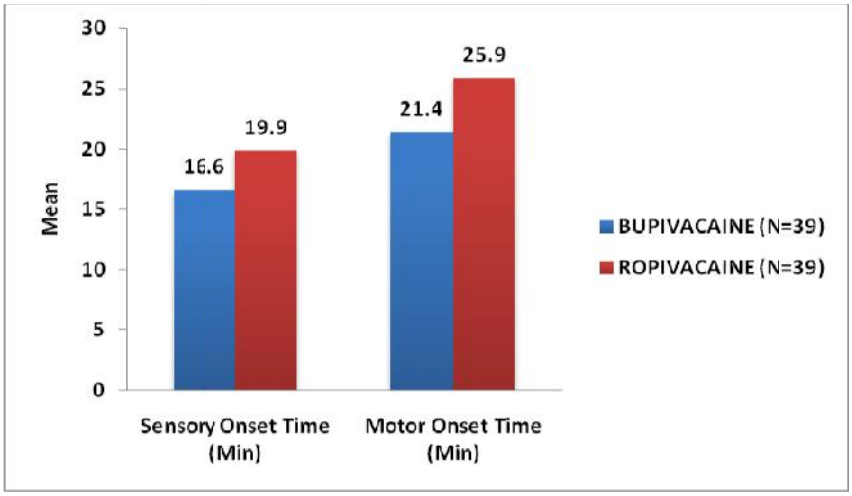


FIGURE - 25: COMPARISON OF ONSET TIME OF SENSORY AND MOTOR BLOCKADE BETWEEN STUDY GROUPS

TABLE 18: COMPARISON OF GROUP B AND GROUP R ON THE BASIS OF DURATION OF SENSORY AND MOTOR BLOCKADE

Parameters	BUPIVACAINE (N=39)		ROPIVACAINE (N=39)		p value
	Mean	SD	Mean	SD	
Duration Sensory blockade (Min)	343.8	44.4	317.9	29.1	0.003
Duration Motor blockade (Min)	387.4	36.0	368.7	33.1	0.019

In Group B , the mean duration of sensory blockade and motor blockade was 343.8 ± 44.4 min and 387.4 ± 36.0 min respectively when compared to Group R having mean duration of sensory blockade and motor blockade of 317.9 ± 29.1 min and 368.7 ± 33.1 min respectively .

Comparison of mean duration time of sensory and motor blockade between the groups

Duration of sensory and motor blockade was prolonged in Group B when compared with Group R . The p value was 0.003 and 0.019 respectively which was statistically highly significant.

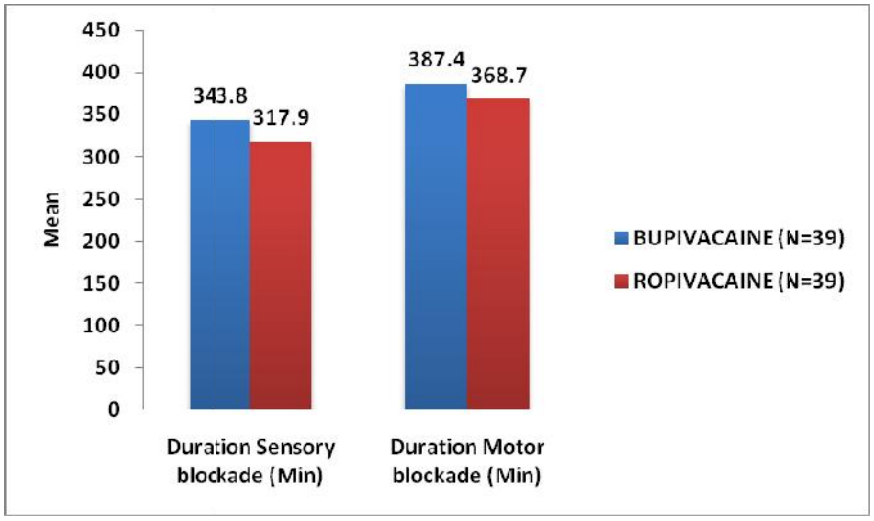


FIGURE – 26: COMPARISION OF DURATION OF SENSORY AND MOTOR BLOCKADE BETWEEN STUDY GROUPS

TABLE 19: ADVERSE EFFECTS/ COMPLICATIONS

Complications	BUPIVACAINE		ROPIVACAINE		Total	p value
	N	Percent	N	Percent		
Nil	37	94.9	39	100	76	0.152
Vomiting	2	5.1	0	0.0	2	
Total	39	100.0	39	100.0	78	

As shown in table 19, the side effects / complication rate are almost negligible if right dose is used and properly deposited.

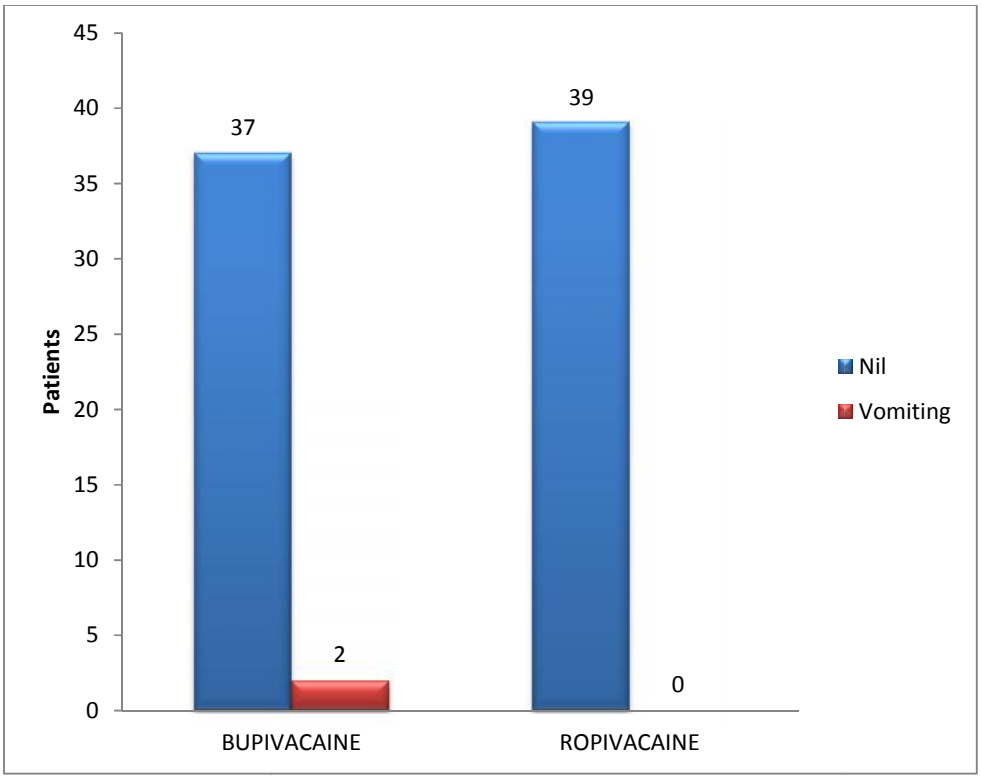


FIGURE – 27: ADVERSE EFFECTS / COMPLICATIONS

DISCUSSION

Brachial plexus block has several clinical applications and has several advantages over general anaesthesia for surgical procedures involving upper extremities. Brachial plexus block is the most commonly performed major peripheral nerve block technique.

The supraclavicular approach to brachial block is carried at the level of trunks of brachial plexus. It provides most effective blockade since plexus is blocked at the middle of brachial plexus, resulting in homogenous spread of anaesthetic drug throughout the plexus. This compactness may explain the block's historical reputation of providing short latency and the most complete and reliable anaesthesia available for upper extremity surgery. Supraclavicular block is often called as "spinal anaesthesia for upper extremity" because of its ubiquitous application for upper extremity surgery characteristically associated with a rapid onset of anaesthesia, high success rate, complete and predictable anaesthesia for upper extremity.

Brachial plexus block with its capability to produce a safe and dense surgical anaesthesia with minimal physiological derangement, has several advantages like reduced pain scores, decreased need for intra operative and postoperative analgesics, rapid recovery and hence shorter hospital stay. It is associated with reduced incidence of intra operative and post operative ischemic events (especially in patients with CAD), attenuation of the metabolic stress response to surgery, decreased incidence of intra operative and postoperative pulmonary complications, decreased blood loss, improved cognitive function with increased activity and improved mobility in post operative period.

Therefore in our study we preferred to use brachial plexus block for patients undergoing upper extremity surgeries. It is a well accepted component of

comprehensive anaesthesia care and of great value particularly in patients who are at high risk for surgery and in emergency situations where patients are full stomach and prone for aspiration. It provides excellent anaesthesia without loss of consciousness and protective airway reflexes.

A significant difference exists between various local anaesthetics like Lignocaine, Mepivacaine, Bupivacaine in terms of onset times, total duration and safety profile when used in brachial blocks. Ropivacaine is a newer long acting amide local anaesthetic found to be equally efficacious to Bupivacaine, but with a better safety profile when used in brachial block.

The study was a randomized comparative study carried out at B.L.D.E.U's Shri B.M. Patil Medical College, Hospital and Research Center, Vijayapur. Seventy eight ASA grade I and ASA grade II patients undergoing elective upper limb surgeries were included in the study. Patients were divided into 2 groups of 39 each (Group B and Group R).

Group B received supraclavicular brachial plexus block with 30 ml of 0.5% Bupivacaine. Group R received supraclavicular Brachial plexus block with 30 ml of 0.5% Ropivacaine. Parameters observed included onset of sensory blockade, onset of motor blockade, duration of sensory blockade, duration of motor blockade, and any adverse effects were noted.

Patient characteristics across the groups

The patients in our study groups did not vary much with respect to age, sex, or weight. The type of surgeries performed were almost identical in both the groups. The study groups did not vary much with respect to duration of surgery (statistically not significant).

Changes in the perioperative cardiovascular parameters

There was no significant differences between the study groups with respect to pattern of changes in heart rate, systolic blood pressure, diastolic blood pressure, SpO_2, respiratory rate perioperatively.

In the study conducted by Tripathi *et al*,[28] in 2012 it was found that hemodynamics remained stable in both the Bupivacaine and Ropivacaine groups for supraclavicular brachial plexus block.

Hence, we conclude that there was no difference between Bupivacaine 0.5 % and Ropivacaine 0.5 % with respect to variation in hemodynamic parameters when used at equal volumes for supraclavicular brachial plexus block.

Onset of sensory block and motor block

In our study, we observed that onset of sensory block was earlier in Bupivacaine group (Group B) having a mean value of 16.6±3.2 minutes in comparison with Ropivacaine group (Group R) having a mean value of 19.9±4.0 minutes, which was statistically significant .

In our study, we observed that onset of motor block was earlier in Bupivacaine group (Group B) having a mean value of 21.4±2.6 minutes in comparison with Ropivacaine group (Group R) having a mean value of 25.9±2.4 minutes which was statistically significant .

In the study conducted by Tripathi *et al*,[28] in 2012 they found that there was considerable delay in establishing the complete motor blockade and sensory blockade with Ropivacaine. In contrast to Ropivacaine, the peak effect of sensory and motor blockade established earlier in Bupivacaine group ($P< 0.05$).

In the study conducted by Narendra babu *et al,*[18] in 2014 the mean onset time of sensory block was earlier in Bupivacaine group, 17.70±2.35 minutes in comparison

with Ropivacaine group, 22.13±0.5 minutes ($p<0.05$). The mean onset time of motor block was earlier in Bupivacaine group, 25.43±2.22 minutes in comparison with Ropivacaine group, 27.90± 1.88 minutes ($P<0.05$).

In the study conducted by Singelyn,[2] in 2001 "Clinical application of Ropivacaine for the upper extremity", found that Ropivacaine is at least as efficient as Bupivacaine in terms of quality, duration of analgesia, anaesthesia and motor block. It could have some advantages over Bupivacaine in terms of onset time of sensory and motor block, but this remains controversial.

In the study conducted by Vaghadia *et al*,[22] in 1999 it was found that Ropivacaine at a concentration of 0.75% (7.5 mg/ml) was required to produce effective and well tolerated brachial plexus block of long duration by the subclavian perivascular route similar to those of 30ml Bupivacaine 0.5%.

The above observations were similar to our study results. Hence, we conclude that Bupivacaine 0.5 % has an advantage of early onset of sensory and motor blockade when compared to Ropivacaine 0.5% for supraclavicular brachial plexus block at equal volume.

Duration of sensory block and motor block

In our study the duration of sensory block was longer in Bupivacaine group (Group B) having a mean value of 343.8±44.4 minutes in comparison with Ropivacaine group (Group R) having a mean value of 317.9±29.1 minutes which was statistically significant.

In our study the duration of motor block was longer in Bupivacaine group (Group B) having a mean value of 387.4±36.0 minutes in comparison with Ropivacaine group (Group R) having a mean value of 368.7±33.1 minutes which was statistically significant.

In the study conducted by Mcglade *et al,*[21] in 1998 comparing 0.5% Ropivacaine and 0.5% Bupivacaine for brachial plexus block they noted that the quality of anaesthesia was similar, however the motor blockade lasted significantly longer when Bupivacaine was used.

In the study conducted by Narendra babu *et al,*[18] in 2014 the duration of sensory block was longer in Bupivacaine group, 342.00±47.66 minutes in comparison with Ropivacine group , 302.00±42.38 minutes ($p<0.05$). The duration of motor block was longer in Bupivacine group, 369.00±41.05 minutes in comparison with Ropivacine group 336.00±37.29 minutes ($p<0.05$).

Reader *et al,*[24] in 1999 in their study "Axillary brachial plexus block with Ropivacaine, a comparative study with Bupivacaine 5mg/ml", showed that 0.75% Ropivacaine used for axillary brachial block resulted in better anaesthesia when compared with same volume of 0.5% Bupivacaine, however the onset and duration of blockade were similar in both groups and concluded that Ropivacaine at a concentration of 7.5 mg/ml was required to produce similar effects with respect to onset and duration of sensory and motor blockade as compared to Bupivacaine 0.5 % at equal volumes .

In the study conducted by McClellan *et al,*[26] in 2000 "Ropivacaine, An update of its use in regional anaesthesia", they concluded that Ropivacaine is a well tolerated regional anaesthetic with an efficacy broadly similar to that of Bupivacaine but has a lower propensity to produce motor blockade. However, it may be a preferred option because of its reduced central nervous system and cardiotoxic potential.

The above observations were similar to our study results. Hence, we conclude that Bupivacaine 0.5 % has an advantage of prolonged duration of sensory blockade

and motor blockade when compared to Ropivacaine 0.5% for supraclavicular brachial plexus block at equal volume.

Adverse effects / complications:

No patient in our study developed any significant side effects.

In our study 2 patients in the Bupivacaine group complained of vomiting compared to none of the patients in Ropivacaine group. This signifies that adverse effects were not significant in both the groups .

In the study conducted by Singelyn,[2] in 2001 "Clinical application of Ropivacaine for the upper extremity" concluded that Ropivacaine is at least as efficient as Bupivacaine in terms of quality, duration of analgesia, anesthesia, and motor block. Because of lower CNS and cardiac toxicity, Ropivacaine is safer than Bupivacaine.

In the study conducted by McClellan *et al*,[26] in 2000 "Ropivacaine an update of its use in regional anaesthesia", they concluded that Ropivacaine is a well tolerated regional anaesthetic with an efficacy broadly similar to that of Bupivacaine. However, it may be a preferred option because of its reduced central nervous system and cardiotoxic potential.

In the study conducted by Vaghadia *et al*,[22] in 1999 to compare efficacy of 30 ml of Ropivacaine 7.5mg/ml with 30 ml Bupivacaine 5mg/ml and 30 ml of Ropivacaine 0.5 % for supraclavicular brachial plexus block, the authors suggested that the lower CNS toxicity and cardiotoxicity of Ropivacaine reduces the risk to the patient due to inadvertent intravenous injection.

We conclude that the side effects/complication rate are almost negligible with both Bupivacaine and Ropivacaine if right doses are used and properly deposited avoiding intravascular injection.

CONCLUSION

On the basis of our study, we can draw the conclusion that at equal volumes Inj.Bupivacaine 0.5% has an advantage over Inj.Ropivacaine 0.5% for supraclavicular brachial plexus block in terms of:

- Early onset of sensory blockade.
- Early onset of motor blockade.
- Prolonged duration of sensory blockade.
- Prolonged duration of motor blockade.
- Both the drugs maintain stable hemodynamic profile perioperatively and adverse effects/complication rate are almost negligible if right doses are used and properly deposited, avoiding intravascular injection.

SUMMARY

The present study entitled “A comparative study of inj.Bupivacine 0.5% and inj.Ropivacine 0.5% for supraclavicular brachial plexus block was carried out in the Department of Anaesthesiology, B.L.D.E. University’s Shri B M Patil Medical College Hospital and Research Centre, Vijayapur. Seventy eight ASA grade I and ASA grade II patients undergoing elective upper limb orthopedic surgeries were included in the study.

Patients were divided into 2 groups of 39 each (Group B and Group R). Group B received supraclavicular brachial plexus block with 30 ml of 0.5% Bupivacaine. Group R received supraclavicular brachial plexus block with 30 ml of 0.5% Ropivacaine. Parameters observed included onset of sensory blockade, onset of motor blockade, duration of sensory blockade, duration of motor blockade and any adverse effects /complications were noted.

Under aseptic precautions, all the patients received supraclavicular brachial plexus block by ultrasound guided approach. All necessary equipments and drugs needed for administration of general anaesthesia were kept ready in order to manage failure of the block.

The patients in our study groups did not vary much with respect to age, sex or weight. The type of surgeries performed were almost identical in both the groups. The study groups did not vary much with respect to duration of surgery (statistically not significant).

There was no significant differences between the study groups with respect to pattern of changes in heart rate, systolic blood pressure, diastolic blood pressure, SpO_2, respiratory rate perioperatively.

Onset of sensory blockade was earlier in Group B (Bupivacaine) having a mean value of 16.6±3.2 minutes in comparison with Group R (Ropivacaine) having a mean value of 19.9±4.0 minutes which was statistically significant. Duration of sensory blockade was also longer in Group B (Bupivacaine) having a mean value of 343.8±44.4 minutes in comparison with Group R (Ropivacaine) having a mean value of 317.9±29.1 minutes which was statistically significant .

Onset of motor blockade was earlier in Group B (Bupivacaine) having a mean value of 21.4±2.6 minutes in comparison with Group R (Ropivacaine) having a mean value of 25.9±2.4 minutes which was statistically significant. Duration of motor blockade was also longer in Group B (Bupivacaine) having a mean value of 387.4±36.0 minutes in comparison with Group R (Ropivacaine) having a mean value of 368.7±33.1 minutes which was statistically significant.

With the present study we can summarize that Inj.Bupivacaine 0.5 % has early onset of sensory blockade, early onset of motor blockade, prolonged duration of sensory blockade, prolonged duration of motor blockade, when compared to Inj.Ropivacaine 0.5 % at equal volumes. Both the drugs maintain stable hemodynamic profile perioperatively and are devoid of any adverse effects at the concentration and volumes used for the study.

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ANNEXURES

ETHICAL CLEARANCE CERTIFICATE

B.L.D.E. UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103
INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 13-11-2013 *at* 3-30pm *to scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis has been accorded Ethical Clearance.*

Title "A Comparative Study of Inj. Bupivacaine 0.5% and Inj. Ropivacaine 0.5% for Supraclavicular brachial plexus block"

Name of P.G. student Dr. Shishir K.R.
Department of Anaesthesiology

Name of Guide/Co-investigator Dr Vijay V. Katti
Associate Professor, Anaesthesiology

DR.TEJASWINI. VALLABHA
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Following documents were placed before E.C. for Scrutinization
1) Copy of Synopsis/Research project.
2) Copy of informed consent form
3) Any other relevant documents.

PROFORMA

A COMPARATIVE STUDY OF INJ.BUPIVACAINE 0.5% AND INJ.ROPIVACAINE 0.5% FOR SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK

Name:

Age: Date :

Sex: IP No. :

PRE-OPERATIVE EVALUATION :

GPE: Respiratory Rate:

Pulse rate: Temperature:

Blood Pressure: Weight:

SYSTEMIC EXAMINATION :

CVS: Mallampatti grading of airway:

RS:

CNS:

Others:

INVESTIGATIONS :

Hb%: Urine:

BT: HIV:

CT: HbsAg:

RBS: ECG:

Blood Urea : Serum Creatinine:

PRE-OPERATIVE DIAGNOSIS :

PROPOSED SURGERY :

ASA grade :

ANAESTHETIC TECHNIQUE:

Supraclavicular approach to brachial plexus block by ultrasound guided technique.

GROUPS:

1) Group B : Bupivacaine group receive 30ml, Bupivacaine 0.5% (5mg/ml)

2) Group R : Ropivacaine group receive 30ml, Ropivacaine 0.5%, (5mg/ml)

DURATION OF SURGERY:

OBJECTIVES:

1. Time of Injection: _____min.
2. Time of onset of sensory Blockade: _____min.
3. Time of onset of motor Blockade: _____min.
4. Duration of sensory Blockade: _____min.
5. Duration of motor Blockade: _____min.
6. Any Adverse effects : _____.

MONITORING :

TIME	SpO_2 (%)	RR (Per min)	BP (mm of Hg)	Heart Rate (Per min)
Pre-Operative				
5 min. after blockade				
15 min. after blockade				
30 min. after blockade				
60 min. after blockade				
2 hrs. after blockade				
3 hrs. after blockade				
6 hrs. after blockade				
12 hrs. after blockade				

DATE: STAFF SIGNATURE.

SAMPLE INFORMED CONSENT FORM

B.L.D.E.U.'s SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR – 586103, KARNATAKA

TITLE OF PROJECT: "A COMPARATIVE STUDY OF INJ.BUPIVACAINE 0.5% AND INJ.ROPIVACAINE 0.5% FOR SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK "

PRINCIPAL INVESTIGATOR : DR. SHISHIR K.R
Department of Anaesthesiology
Email: shishirsmashes@yahoo.co.in

PG GUIDE: **DR. VIJAY.V.KATTI**
ASSOCIATE PROFESSOR
DEPARTMENT OF ANAESTHESIOLOGY
B.L.D.E.U.'s SHRI B.M. PATIL MEDICAL COLLEGE ,VIJAYAPUR.

PURPOSE OF RESEARCH:

I have been informed that this study is **"A COMPARATIVE STUDY OF INJ.BUPIVACAINE 0.5% AND INJ.ROPIVACAINE 0.5% FOR SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK "** I have been explained about the reason for doing this study and selecting me/my ward as a subject for this study. I have also been given free choice for either being included or not in the study.

PROCEDURE:

I understand that I will be doing **"A COMPARATIVE STUDY OF INJ.BUPIVACAINE 0.5% AND INJ.ROPIVACAINE 0.5% FOR SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK**

RISKS AND DISCOMFORTS:

I understand that I/my ward may experience some pain while giving supraclavicular brachial plexus block and I understand that necessary measures will be taken to reduce these complications as and when they arise.

BENEFITS:

I understand that my/my wards participation in this study will help in finding out **"A COMPARATIVE STUDY OF INJ.BUPIVACAINE 0.5% AND INJ.ROPIVACAINE 0.5% FOR SUPRACLAVICULAR BRACHIAL PLEXUS BLOCK"**

CONFIDENTIALITY:

I understand that medical information produced by this study will become a part of this Hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of the medical records, but will be stored in the investigator's research file and identified only by a code number. The code key connecting name to numbers will be kept in a separate secure location.

If the data are used for publication in the medical literature or for teaching purpose, no names will be used and other identifiers such as photographs and audio or video tapes will be used only with my special written permission. I understand that I may see the photograph and videotapes and hear audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time**.**

Dr. Shishir K.R. is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation.

If during this study, or later, I wish to discuss my participation in or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me.

And that a copy of this consent form will be given to me for keep for careful reading.

REFUSAL OR WITHDRAWL OF PARTICIPATION:

I understand that my participation is voluntary and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital.

I also understand that Dr. Shishir K.R. will terminate my participation in this study at any time after he has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist, if this is appropriate

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have explained to _____ the purpose of this research, the procedures required and the possible risks and benefits, to the best of my ability in patient's own language.

Date: **DR. VIJAY.V.KATTI** **DR. SHISHIR K. R**

(Guide) (Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that Dr. Shishir K.R. has explained to me the purpose of this research, the study procedure that I will undergo and the possible discomforts and benefits that I may experience, in my own language.

I have been explained all the above in detail in my own language and I understand the same. Therefore I agree to give my consent to participate as a subject in this research project.

_____ _____

(Participant) Date

_____ _____

(Witness to above signature) Date

KEY TO THE MASTER CHART

BB ……..	Both bone
RT ……...	Right
LT……	Left
CR……	Closed reduction
ORIF……	Open reduction internal fixation
CRIF……	Closed reduction internal fixation
DCP…….	Dynamic compression plating
TBW…….	Tension Band wiring
#.....	Fracture

MASTER CHART

										Onset Time (Min)		Duration (min)		Heart rate (per min)									Systolic BP (mm of Hg)									Diastolic BP (mm of Hg)									
Group	S.No	Name	IP No.	Age (yrs)	Sex	Weight (kg)	Diagnosis	Surgery	Duration of Surgery (Min)	Sensory	Motor	Sensory blockade	Motor blockade	0 min	5 min	15 min	30 min	60 min	2 hrs	3 hrs	6 hrs	12 hrs	0 min	5 min	15 min	30 min	60 min	2 hrs	3 hrs	6 hrs	12 hrs	0 min	5 min	15 min	30 min	60 min	2 hrs	3 hrs	6 hrs	12 hrs	Complications
GROUP B : BUPIVACAINE GROUP																																								
GroupB	1	ANAND	975	26	M	72	# LT DISTAL END RADIUS	ORIF & TBW,K WIRING	**90**	15	19	280	360	96	79	84	87	81	94	88	92	85	122	120	118	117	116	114	113	112	110	76	75	73	72	70	70	70	70	73	Nil
GroupB	2	POOJA	3081	38	F	68	# RT BB FOREARM	ORIF& DCP PLATE	120	13	22	330	430	82	97	97	90	93	87	97	84	83	127	122	117	115	114	112	111	110	108	80	79	77	76	70	70	70	70	71	Nil
GroupB	3	NINGANGOUDA	35744	31	M	74	#DISTAL HUMERUS	ORIF& DCP PLATE	140	18	20	280	340	99	100	92	91	90	92	88	82	100	121	121	118	115	114	112	111	110	108	73	72	72	71	70	73	70	70	72	Nil
GroupB	4	MADAPPA	1338	52	M	54	#RT DISTAL END RADIUS	ORIF& DCP PLATE	130	14	21	390	400	84	84	96	99	95	100	90	99	81	123	122	118	118	117	115	114	113	111	80	79	77	76	71	73	70	71	70	Nil
GroupB	5	MANIKANTH	2578	36	M	69	# RT DISTAL END RADIUS & ULNA	ORIF& DCP PLATE	160	19	22	370	380	80	93	99	85	83	100	80	88	91	122	122	122	117	116	114	113	112	110	80	79	77	76	72	70	71	70	73	Nil
GroupB	6	RENUKA	4993	42	F	73	# RT COLLES	CRIF& K-WIRING	150	13	20	360	440	98	87	95	84	83	96	93	100	88	123	122	121	115	114	112	111	110	108	77	76	74	73	70	72	70	70	70	Nil
GroupB	7	LALUSAB	8522	51	M	78	# LT SUPRACONDYLAR HUMERUS	ORIF& DCP PLATE	60	18	24	280	340	80	98	82	83	91	85	80	83	87	126	122	121	117	116	114	113	112	110	76	75	73	72	70	71	70	70	72	Nil
GroupB	8	RENUKA	117787	41	F	68	# LT ELBOW DISLOCATION	CRIF & K-WIRING	70	18	22	320	440	91	81	96	90	90	83	99	80	85	121	121	116	115	114	112	111	110	108	80	79	77	76	73	71	70	71	72	vomiting
GroupB	9	GURUPAD	12760	40	M	78	# LT BB FOREARM NON UNION	DARRACH'S OSTEOTOMY	160	15	20	330	370	97	94	82	91	89	87	95	83	91	126	121	120	117	116	114	113	112	110	80	79	77	76	76	73	73	71	71	Nil
GroupB	10	BASAVARAJ	14161	37	M	66	# LT DISTAL END RADIUS	CRIF & K-WIRING	130	14	23	380	410	94	89	91	85	85	87	80	97	96	121	121	118	117	116	114	113	112	110	80	79	77	76	73	70	70	70	70	Nil
GroupB	11	SURESH	15144	52	M	74	# LT SUPRACONDYLAR HUMERUS	ORIF & DCP PLATE	150	14	22	300	340	89	82	90	83	89	80	79	92	83	126	121	119	117	116	114	113	112	110	74	73	71	70	70	72	70	71	71	Nil
GroupB	12	VINOTH	16406	28	M	72	# LT SHAFT OF RADIUS	CRIF & K WIRING	140	15	22	310	440	87	83	80	94	96	96	94	92	91	124	122	117	116	115	113	112	111	109	73	72	72	71	70	70	70	70	70	Nil
GroupB	13	SHEKAR	17321	42	M	68	# RT COLLES	CRIF & K WIRING	110	15	21	380	390	95	81	99	85	86	83	87	80	84	123	121	121	118	117	115	114	113	111	77	76	74	73	70	70	70	70	71	Nil
GroupB	14	SHANKAR	17464	35	M	74	# RT RADIUS & # FIRST METACARPAL	ORIF& DCP PLATE & K WIRING	130	15	19	310	390	93	99	95	80	89	87	84	92	80	124	124	119	115	114	112	111	110	108	80	79	77	76	72	72	70	70	71	Nil
GroupB	15	SHAKAWWA	17500	18	F	54	# RT BB FOREARM IMPLANT INSITU	IMPLANT REMOVAL	150	16	21	280	400	99	80	98	87	97	82	92	100	100	126	122	118	117	116	114	113	112	110	80	79	77	76	73	71	72	72	70	Nil
GroupB	16	CHANDRAN	18818	35	M	69	# LT DISTAL ULNA	ORIF& DCP PLATE	180	25	26	280	340	88	81	83	88	79	94	82	82	93	126	124	119	116	115	113	112	111	109	80	79	77	76	75	73	71	72	70	Nil
GroupB	17	TANGEWWA	18932	55	F	73	# RT BB FOREARM	ORIF& DCP PLATE	110	18	21	300	380	80	84	80	81	93	96	91	86	80	125	124	119	117	116	114	113	112	110	79	78	76	75	75	71	71	71	72	Nil
GroupB	18	SUMAN	19013	37	M	78	# RT COLLES & #FIRST METACARPAL	ORIF& DCP PLATE & K WIRING	100	25	30	320	330	99	96	82	99	99	97	100	92	99	125	124	121	117	116	114	113	112	110	80	79	77	76	75	73	71	71	71	Nil
GroupB	19	KAILASH	18991	46	M	53	# RT SHAFT ULNA	ORIF& DCP PLATE	160	16	19	290	330	93	86	98	84	87	87	85	85	97	120	120	120	117	116	114	113	112	110	80	79	77	76	74	72	70	72	70	Nil
GroupB	20	CHANDRAM	18818	35	M	66	#BB FOREARM NON UNION	DARRACH'S OSTEOTOMY	100	24	27	350	360	85	85	87	98	90	95	93	89	79	126	122	118	116	115	113	112	111	109	79	78	76	75	74	70	70	70	73	Nil
GroupB	21	SUREKHA JADHAV	20796	38	F	66	#RT COLLES	CR & CAST APPLICATION	140	14	18	350	360	94	86	96	95	81	93	86	91	100	124	120	120	117	116	114	113	112	110	79	78	76	75	75	70	70	71	73	Nil
GroupB	22	CHIDANAND	20871	35	M	72	# SHAFT OF ULNA	ORIF& DCP PLATE	140	19	21	300	420	81	90	81	99	79	99	91	88	96	127	120	118	116	115	113	112	111	109	76	75	73	72	72	73	71	71	70	Nil
GroupB	23	LALITHA	233293	46	F	66	RT ELBOW DISLOCATION	CRIF & K WIRING	90	18	19	410	420	86	88	94	96	87	93	88	95	93	125	122	116	116	115	113	112	111	109	80	79	77	76	70	70	72	70	71	Nil
GroupB	24	MANOJ DAVANGIRI	21568	55	M	73	# RT DISTAL END RADIUS	ORIF & K WIRING	110	15	20	350	360	96	80	91	96	90	82	86	82	82	122	122	122	115	114	112	111	110	108	77	76	74	73	70	70	70	72	73	Nil
GroupB	25	VIDYA MALLAWWA	21362	40	F	64	# RT SUPRACONDYLAR HUMERUS	ORIF& DCP PLATE	110	19	21	280	340	92	81	98	99	99	93	85	95	93	125	124	122	117	116	114	113	112	110	74	73	71	70	70	70	70	70	71	Nil
GroupB	26	RAJASHEKAR	26425	39	M	76	# LT BB FOREARM	ORIF& DCP PLATE	120	15	18	410	420	84	92	93	98	98	88	96	83	80	122	121	119	115	114	112	111	110	108	73	72	72	71	70	70	70	70	73	Nil
GroupB	27	RAKESH	26815	20	M	68	# LT DISTAL RADIUS IMPLANT INSITU	IMPLANT REMOVAL	150	17	22	390	400	100	94	93	90	83	85	100	100	86	122	120	120	118	117	115	114	113	111	76	75	73	72	70	70	70	70	70	Nil
GroupB	28	PREMA HANUMANTH	26810	25	F	72	# LT COLLES	CRIF & K-WIRING	180	18	22	400	410	83	92	92	82	91	93	83	82	99	125	121	120	118	117	115	114	113	111	78	77	75	74	70	70	71	71	71	Nil
GroupB	29	RENUKA	2185	42	F	68	# RT SUPRACONDYLAR HUMERUS	CRIF & K-WIRING	160	13	17	410	420	91	83	100	94	89	93	86	86	89	124	123	120	117	116	114	113	112	110	80	79	77	76	72	70	72	70	73	vomiting
GroupB	30	NIMBANNA	2986	29	M	60	# LT BB FOREARM	CRIF & K-WIRING	180	17	23	410	420	92	100	90	100	100	95	99	85	86	126	124	120	118	117	115	114	113	111	80	79	77	76	76	72	70	71	71	Nil
GroupB	31	PRAKASH	32625	25	M	72	# RT ULNA IMPLANT INSITU	IMPLANT REMOVAL	100	20	21	370	430	89	94	80	87	95	100	94	86	91	123	123	122	117	116	114	113	112	110	76	75	73	72	70	72	72	70	72	Nil
GroupB	32	RAMAN	3785	45	M	54	# LT BB FOREARM	ORIF & DCP PLATE	80	15	20	360	370	92	86	88	88	81	95	97	81	83	124	123	121	115	114	112	111	110	108	80	79	77	76	72	71	70	70	73	Nil
GroupB	33	ASHA	43630	34	F	74	# RT COLLES	ORIF& K-WIRING	80	15	20	390	400	80	82	88	94	82	85	96	85	89	124	123	121	118	117	115	114	113	111	73	72	72	71	70	71	72	71	70	Nil
GroupB	34	RAKESH	55841	24	M	68	# RT ULNA STYLOID PROCESS	CRIF & K-WIRING	180	19	22	310	340	89	81	92	94	94	93	83	91	84	125	124	117	117	116	114	113	112	110	80	79	77	76	73	70	71	73	71	Nil
GroupB	35	GIRIJA	4944	20	F	56	# LT DISTAL END RADIUS	ORIF & K- WIRING	160	15	26	350	440	84	85	79	85	79	94	100	88	98	127	123	118	116	115	113	112	111	109	80	79	77	76	72	70	70	72	72	Nil
GroupB	36	SHIVALINGAPPA	6745	19	M	68	RT HAND GANGLION	EXCISION	130	13	19	410	440	81	99	83	93	83	84	97	93	98	126	124	120	118	117	115	114	113	111	80	79	77	76	70	73	71	70	72	Nil
GroupB	37	SURESH	7517	30	M	57	# MID-SHAFT RADIUS	EXTERNAL FIXATOR APPLICATION	90	13	23	350	370	79	100	99	95	87	99	79	97	87	122	122	117	116	115	113	112	111	109	80	79	77	76	71	73	71	72	72	Nil
GroupB	38	KAVYA	119622	19	F	58	# LT BB FOREARM & FIRST METACARPAL	ORIF& DCP PLATE & K WIRING	90	19	20	370	380	94	85	89	97	91	88	88	79	83	123	120	120	118	117	115	114	113	111	80	79	77	76	70	70	73	72	73	Nil
GroupB	39	SHIVANAND	11160	53	M	66	# RT SUPRACONDYLAR HUMERUS	ORIF & K-WIRING	100	13	20	350	360	87	84	98	85	79	90	82	85	89	125	124	117	116	115	113	112	111	109	80	79	77	76	75	73	73	71	73	Nil

											Onset Time (Min)		Duration (min)		Heart rate (per min)								Systolic BP (mm of Hg)									Diastolic BP (mm of Hg)									
Group	S.No	Name	IP No.	Age (yrs)	Sex	wt (kg)	Diagnosis	Surgery	Duration of Surgery (Min)	Sensory	Motor	Sensory blockade	Motor blockade	0 min	5 min	15 min	30 min	60 min	2 hrs	3 hrs	6 hrs	12 hrs	0 min	5 min	15 min	30 min	60 min	2 hrs	3 hrs	6 hrs	12 hrs	0 min	5 min	15 min	30 min	60 min	2 hrs	3 hrs	6 hrs	12 hrs	Complications
GROUP R : ROPIVACAINE GROUP																																								
GroupR	1	GANGA	11162	39	F	70	# RT COLLES	CRIF & K-WIRING	110	24	25	300	350	85	88	90	95	100	82	79	79	94	125	124	117	116	115	113	112	111	109	80	79	77	76	74	70	70	70	72	Nil
GroupR	2	SANGAMESH	9955	46	M	58	# RT BB FOREARM	ORIF& DCP PLATE	160	19	22	330	350	94	81	93	99	94	94	87	91	92	124	121	120	115	114	112	111	110	108	79	78	76	75	71	72	71	71	70	Nil
GroupR	3	NINGAMMA	12332	42	F	63	# LT BB FOREARM	ORIF& DCP PLATE	130	20	29	320	370	97	81	92	96	100	82	83	88	96	123	120	119	116	115	113	112	111	109	79	78	76	75	70	72	72	70	71	Nil
GroupR	4	SHANTANU	12442	42	M	68	# RT SUPRACONDYLAR HUMERUS	ORIF & K-WIRING	170	22	26	310	350	97	85	85	83	85	81	87	92	93	126	124	122	117	116	114	113	112	110	80	79	77	76	70	71	70	70	70	Nil
GroupR	5	SARITA	15076	25	F	66	# LT BB FOREARM NON UNION	DARRACH'S OSTEOTOMY	160	22	26	380	390	97	82	96	81	79	83	84	97	94	127	121	119	117	116	114	113	112	110	80	79	77	76	74	71	72	72	72	Nil
GroupR	6	BASAVARAJ	14811	36	M	58	# RT RADIUS	ORIF & K-WIRING	170	22	29	290	350	79	88	89	92	86	93	92	79	97	123	120	118	115	114	112	111	110	108	77	76	74	73	73	70	70	70	73	Nil
GroupR	7	PARAMANNA	176532	48	M	66	LT ELBOW DISLOCATION	CR & CAST APPLICATION	140	16	27	360	430	84	89	92	98	84	100	81	83	85	126	124	122	116	115	113	112	111	109	78	77	75	74	70	71	70	70	70	Nil
GroupR	8	SHASHIDHAR	180144	28	M	74	# RT SHAFT RADIUS	CRIF & K WIRING	100	14	23	300	340	88	94	99	88	87	98	84	93	100	125	124	121	115	114	112	111	110	108	80	79	77	76	76	70	70	72	73	Nil
GroupR	9	SUSHILABAI	130057	30	F	66	# LT COLLES	ORIF & K WIRING	180	25	29	320	330	99	91	85	88	98	93	88	91	92	125	121	120	118	117	115	114	113	111	80	79	77	76	73	70	70	70	73	Nil
GroupR	10	GIRISH	17036	42	M	74	# LT BB FOREARM IMPLANT INSITU	IMPLANT REMOVAL	110	14	23	350	370	97	89	94	79	91	87	85	92	94	120	120	120	117	116	114	113	112	110	79	78	76	75	74	73	72	70	70	Nil
GroupR	11	HANUMANTH	17829	54	M	72	# RT COLLES	CRIF& DCP PLATE	140	21	25	300	350	95	99	85	81	97	82	80	97	99	123	122	118	116	115	113	112	111	109	79	78	76	75	72	73	72	71	71	Nil
GroupR	12	HEMA YALAVAN	17807	23	F	56	# LT COLLES	CRIF& K-WIRING	90	24	30	300	350	94	97	84	91	84	85	82	95	98	122	122	120	116	115	113	112	111	109	76	75	73	72	72	70	70	73	72	Nil
GroupR	13	VEERESH	115403	24	M	70	# LT ELBOW DISLOCATION	CR & CAST APPLICATION	100	23	27	330	350	99	85	82	85	84	86	94	88	96	127	124	121	116	115	113	112	111	109	80	79	77	76	76	72	71	70	70	Nil
GroupR	14	BASAVARAJ	193536	34	M	68	#TENDON INJURY FOREARM	TENDON REPAIR	160	14	25	370	380	90	86	83	84	99	96	83	92	95	122	122	120	118	117	115	114	113	111	80	79	77	76	71	70	72	73	73	Nil
GroupR	15	RAJESHWARI	18246	28	F	78	# LT SHAFT OF ULNA	ORIF& DCP PLATE	170	23	29	370	430	92	92	85	87	97	81	91	97	91	124	123	117	115	114	112	111	110	108	80	79	77	76	76	72	72	72	73	Nil
GroupR	16	SUMAN	19013	37	M	68	# RT RADIAL STYLOID PROCESS	CRIF & K WIRING	160	21	28	330	440	85	85	98	90	98	100	91	98	100	123	123	117	115	114	112	111	110	108	80	79	77	76	75	72	70	72	70	Nil
GroupR	17	JAKRIAPPA	20002	48	M	78	# BB FOREARM IMPLANT INSITU	IMPLANT REMOVAL	130	22	27	300	430	80	85	99	95	81	100	86	84	100	121	120	119	118	117	115	114	113	111	80	79	77	76	74	70	72	73	73	Nil
GroupR	18	HAMEED	20244	42	M	66	# RT DISTAL END RADIUS	ORIF& DCP PLATE	80	19	23	380	400	99	95	81	92	80	90	82	82	84	124	122	118	117	116	114	113	112	110	74	73	71	70	70	71	70	70	73	Nil
GroupR	19	RAVIKUMAR	20374	45	M	74	# LT COLLES	CR & CAST APPLICATION	80	14	22	310	440	80	88	97	81	99	89	80	90	84	122	122	120	115	114	112	111	110	108	78	77	75	74	73	70	71	72	72	Nil
GroupR	20	SIDDALINGAPPA	20484	50	M	72	# LT DISTAL END RADIUS	ORIF& DCP PLATE	80	17	22	340	350	80	85	93	97	87	92	98	89	90	127	124	120	118	117	115	114	113	111	78	77	75	74	74	71	70	70	70	Nil
GroupR	21	MOHAN	247291	50	M	68	# LT BB FOREARM	EXTERNAL FIXATOR APPLICATION	80	18	26	280	390	79	82	94	80	100	93	79	98	100	126	122	118	117	116	114	113	112	110	79	78	76	75	74	70	70	70	72	Nil
GroupR	22	YASEEN MAHABOOB	22348	35	M	74	# RT DISTAL END RADIUS	ORIF & K-WIRING	80	23	25	300	340	86	89	85	82	96	93	97	88	100	127	122	117	115	114	112	111	110	108	78	77	75	74	74	73	70	70	71	Nil
GroupR	23	MADHU	269617	45	M	54	# RT ELBOW DISLOCATION	CR & CAST APPLICATION	80	30	24	280	320	81	94	91	80	82	97	96	97	98	124	124	116	115	114	112	111	110	108	79	78	76	75	74	73	70	72	70	Nil
GroupR	24	CHANNAMMA	25033	35	F	69	# LT OLECRENON	CC SCREW FIXATION& K WIRING	80	21	25	300	380	99	88	89	79	85	98	93	89	79	123	122	122	118	117	115	114	113	111	78	77	75	74	72	71	70	70	70	Nil
GroupR	25	KALLAWWA	25644	32	F	73	GANGLION OVER RT WRIST JOINT	EXCISION	80	19	27	370	410	100	82	94	100	83	94	97	92	98	124	121	117	116	115	113	112	111	109	76	75	73	72	70	71	70	70	72	Nil
GroupR	26	SANTOSH	26131	45	M	78	# RT RADIAL STYLOID PROCESS	ORIF& K-WIRING	80	25	26	290	340	86	91	97	89	86	99	92	82	82	124	120	120	118	117	115	114	113	111	75	74	72	71	70	70	70	70	70	Nil
GroupR	27	MALLAMMA	303672	45	F	53	# LT COLLES	CRIF & K-WIRING	80	13	28	330	360	98	83	96	94	99	79	83	83	100	124	121	119	118	117	115	114	113	111	72	71	71	70	70	70	73	71	70	Nil
GroupR	28	SANDHYA	27362	23	F	66	# LT BB FOREARM	ORIF & DCP PLATE	170	23	22	300	360	86	94	82	97	92	87	81	82	79	126	124	118	118	117	115	114	113	111	73	72	72	71	70	73	71	71	70	Nil
GroupR	29	RENUKA	31073	30	F	66	# RT DISTAL END RADIUS	EXTERNAL FIXATOR APPLICATION	120	24	25	300	350	82	86	97	98	79	80	87	80	80	124	123	122	118	117	115	114	113	111	77	76	74	73	72	70	72	72	72	Nil
GroupR	30	KUMAR	18269	25	M	72	# LT SHAFT OF ULNA	CRIF & K WIRING	170	15	26	350	420	90	79	96	99	84	94	93	89	96	122	122	117	115	114	112	111	110	108	80	79	77	76	72	72	70	73	71	Nil
GroupR	31	SHALINI	1470	35	F	66	# RT SUPRACONDYLAR HUMERUS	CRIF & K-WIRING	80	18	29	310	330	85	93	95	87	100	98	96	98	92	126	124	118	116	115	113	112	111	109	73	72	72	71	70	73	73	72	70	Nil
GroupR	32	ARUN	2212	21	M	73	# LT SUPRACONDYLAR HUMERUS	ORIF & K-WIRING	110	18	29	300	350	83	91	100	80	80	94	93	92	100	122	121	121	115	114	112	111	110	108	72	72	72	71	70	72	72	71	70	Nil
GroupR	33	REVATHI	3929	42	F	64	# RT BB FOREARM	ORIF& DCP PLATE	80	25	22	300	350	84	95	81	96	80	94	87	88	82	122	122	121	117	116	114	113	112	110	80	79	77	76	75	73	71	70	71	Nil
GroupR	34	SUNANDA	47603	38	F	76	# LT COLLES	CRIF & K-WIRING	100	22	24	300	350	87	87	84	90	83	93	88	90	100	125	123	121	116	115	113	112	111	109	79	78	76	75	71	70	70	73	70	Nil
GroupR	35	PRASAD	4342	26	M	68	# LT DISTAL END RADIUS	ORIF& K-WIRING	90	17	25	280	350	87	88	93	81	92	91	92	93	90	124	123	120	117	116	114	113	112	110	74	73	71	70	70	72	70	72	72	Nil
GroupR	36	LALBEE	8157	39	M	72	# LT SHAFT OF ULNA	ORIF& DCP PLATE	80	20	28	290	380	87	80	99	87	86	91	95	82	84	125	122	121	116	115	113	112	111	109	77	76	74	73	73	70	70	70	70	Nil
GroupR	37	SADASHIV	7855	50	M	66	# BB FOREARM NON UNION	DARRACH'S OSTEOTOMY	150	13	27	300	350	92	97	97	86	79	83	81	83	87	124	124	118	117	116	114	113	112	110	78	77	75	74	70	70	70	72	71	Nil
GroupR	38	LAKSHMI BAI	96851	37	F	72	# RT DISTAL END RADIUS	CR & CAST APPLICATION	70	20	25	300	350	93	96	97	82	82	87	98	95	79	127	124	122	118	117	115	114	113	111	80	79	77	76	75	73	72	70	72	Nil
GroupR	39	ARAVIND	10554	26	M	66	# RT BB FOREARM & FIRST METACARPAL	ORIF& DCP PLATE & K WIRING	130	17	30	330	350	95	94	95	87	94	93	88	80	99	123	123	117	116	115	113	112	111	109	80	79	77	76	71	70	70	70	73	Nil